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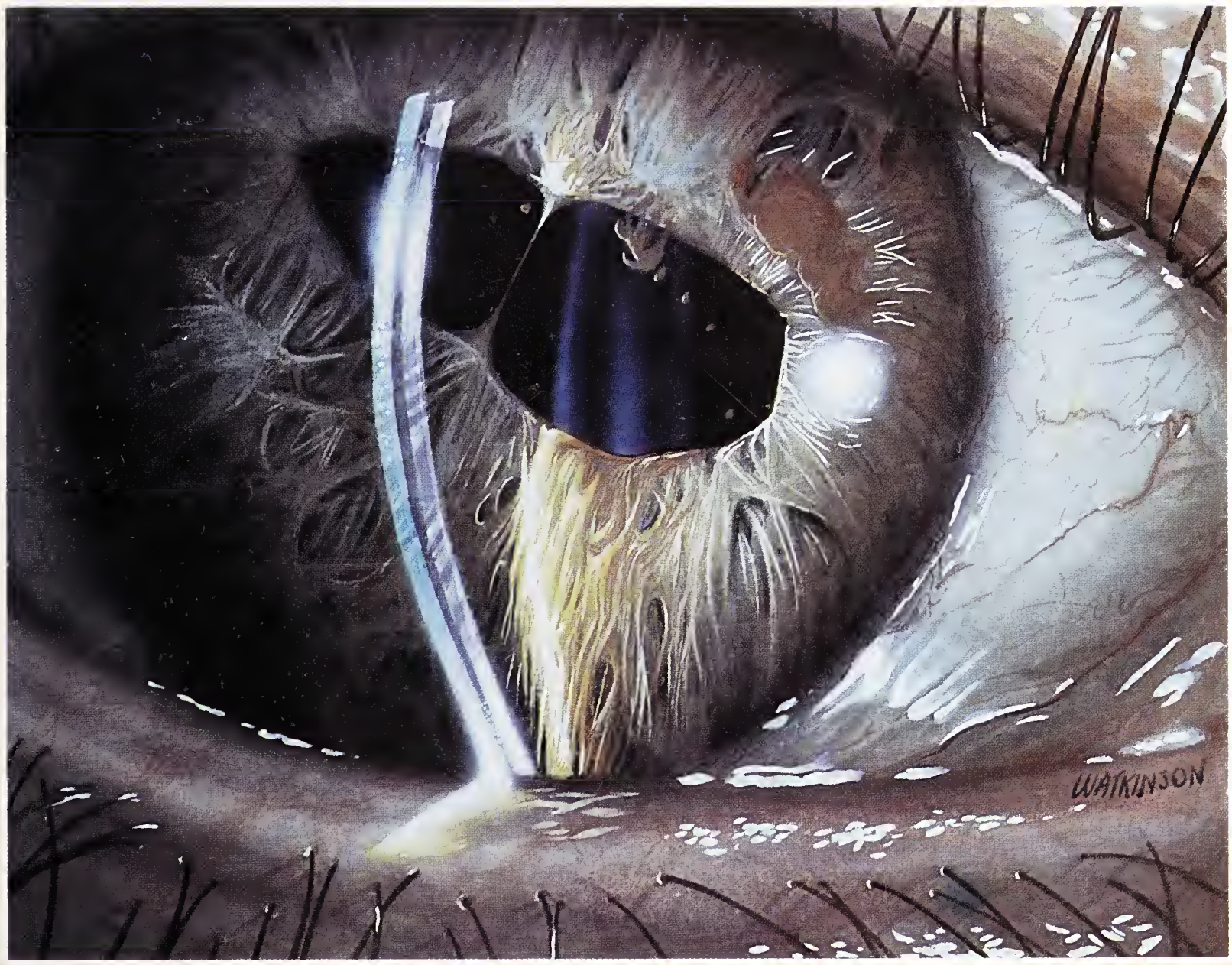
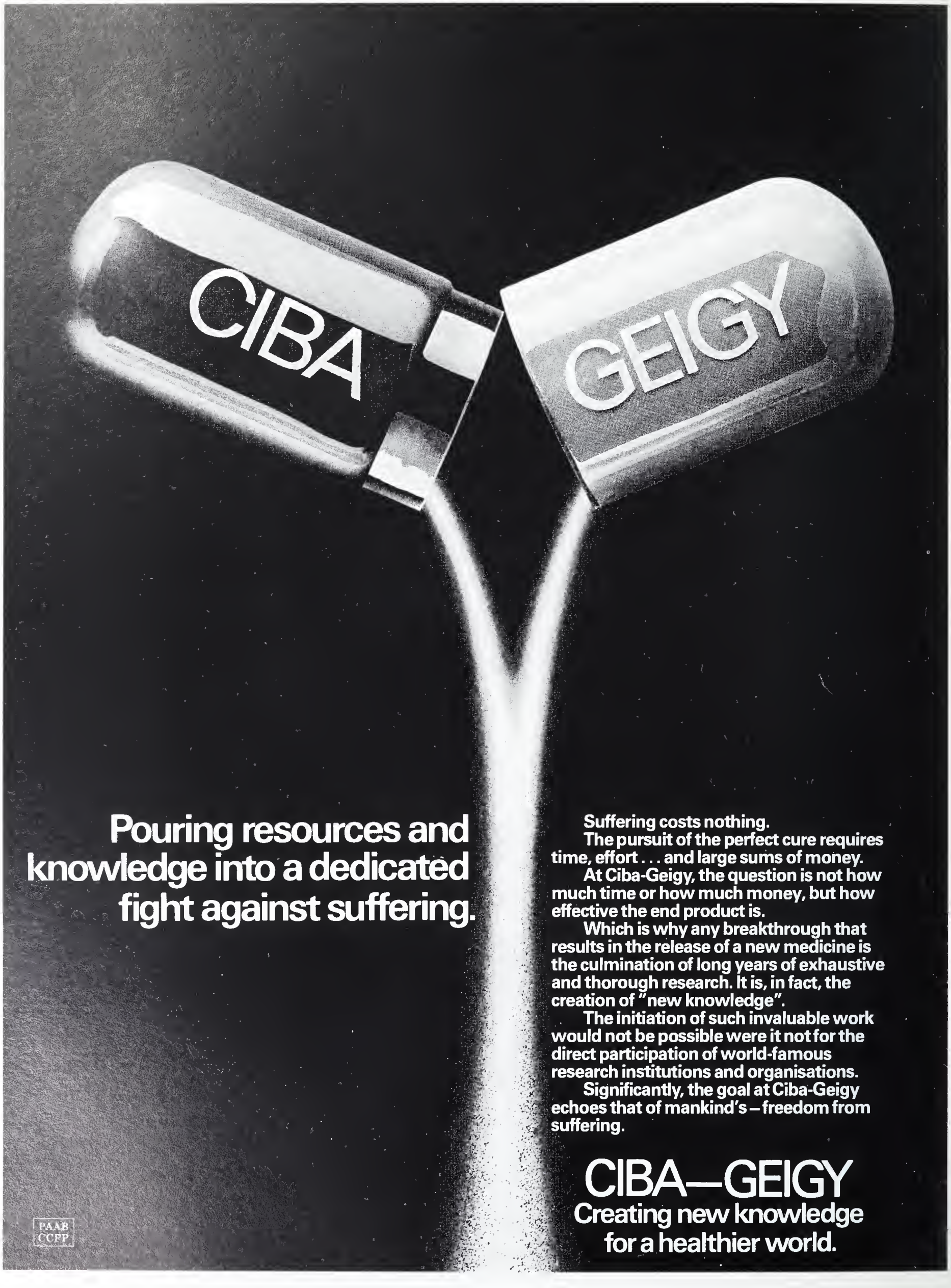


- Blunt Eye Trauma
- Recurrent Shoulder Dislocation
- Prosthetic Cardiac Valves
- Chest X-ray Approach
- Iridocorneal Endothelial Syndrome
- Hereditary Nephritis
- Voice Change

NEW SECTIONS
• Quick Dx
• Impressions



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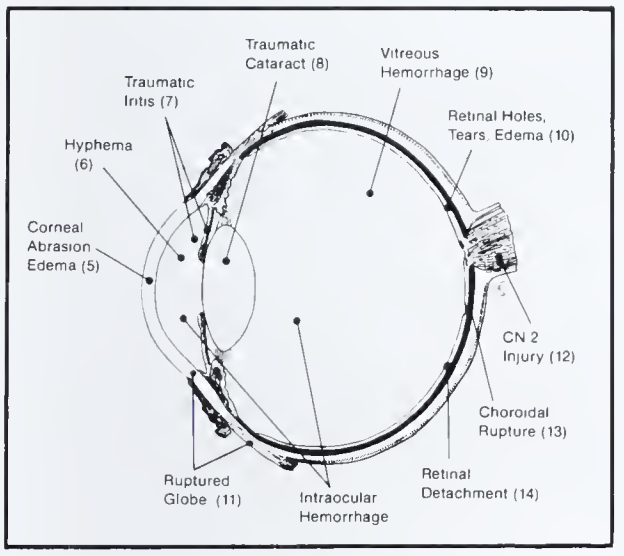

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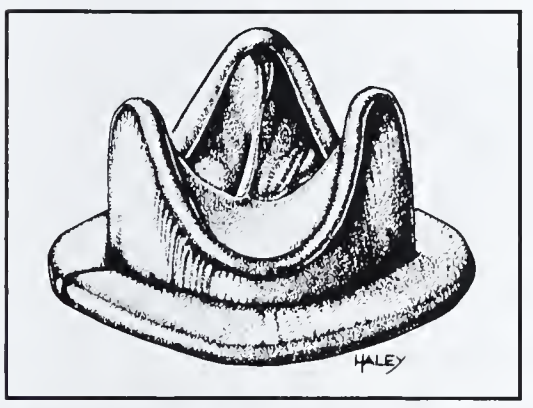

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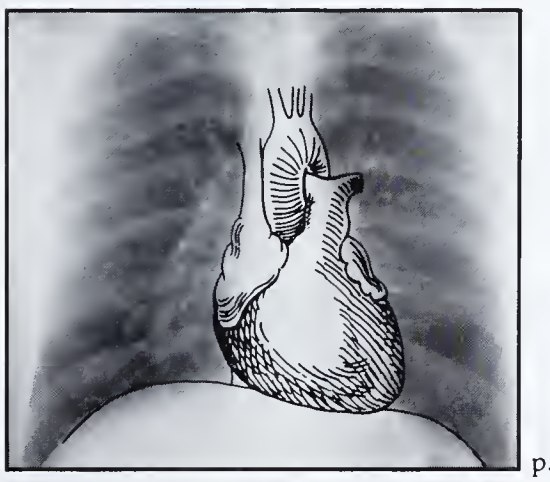

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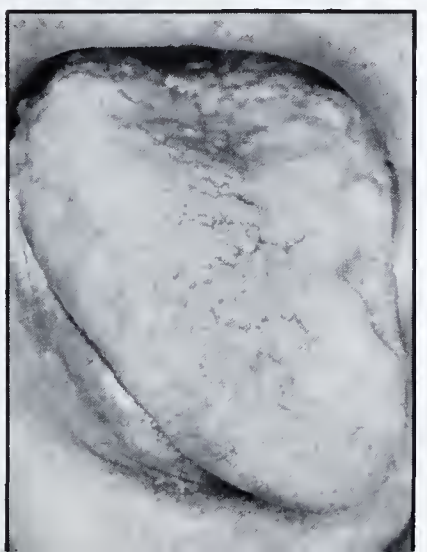

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Cover illustration: Progressive Iris Atrophy by Terry Watkinson. This illustration won the 1988 Award of Excellence from the Canadian Ophthalmological Society.

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Editorial

Michael J. Weinberg
Catherine Birt
Editors-in-Chief

We were very pleased with the first issue and feel that this issue is even more exciting since we have introduced two new sections. The "Dx" section contains some simple sets of problems and answers that the reader can test him/herself with. The types of problem that have been selected are ones that were felt to stress important core knowledge. The second new section, "Impressions", is more literary in nature but still pertains to medicine. The first one in this series is by Ian Park on his experience in Sioux Lookout.

The articles in this issue cover a broad range of topics. There are two articles on ophthalmology. The article on iridiocorneal syndrome is an excellent review of an uncommon yet fascinating condition. The other article is on blunt eye trauma. It stresses an approach using a simple but very powerful concept — first think anatomy; then think pathology. The chest x-ray can be a frightening challenge to those who have to read them, especially for students with little experience in their interpretation. The article describes a simple approach and provides some reference sheets that can be used in the clinic. Animal models for human conditions are often used, have been much criticized but are very useful. A lot can be and has been learned from animal models and hereditary nephritis is a good example of the usefulness of this technique in medical research. Shoulder dislocation is a relatively common orthopaedic condition. The article compares two procedures used to repair recurrent dislocation, in a retrospective study. Finally there is an excellent article on cardiac valves in which the reader learns of the valves used in Toronto. This is important information for the family physician trying to help the patient decide which valve to choose.

We hope that our readers will enjoy this issue. We have tried to be very critical of the articles and have used rigorous editorial standards to ensure both the quality and the readability of the papers in the *Journal*. We also hope that the addition of the two new sections will make the *Journal* more interesting. We wish all our readers the very best in the upcoming year and we thank you for your support.

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B.V.

Diagnosis and Management of Blunt Eye Trauma in Primary Care

Paul Marchetti, 9T0

Abstract

When presented with a case of ocular injury, many physicians will perform a cursory exam before referral to an ophthalmologist. Eye injury, however, is a frequent enough presentation that the examiner should be able to develop a degree of expertise in assessing the patient. Even in multisystem trauma, once life threatening injuries have been stabilized, it is important to ascertain the integrity of the visual system. Whatever the obstacle, ocular, extraocular, and neurological components must be fully assessed. Minor injuries may be treated by the emergency medicine physician, ensuring follow-up for possible complications. Consultation is then appropriate for serious injuries, or to assess any doubts that remain. It is hoped that this succinct schematic will offer the primary care physician a convenient repertoire of common as well as serious manifestations of blunt eye trauma. It may also help assimilate a sound and logical examination of the visual system.

Children might suffer blunt eye trauma from a poke by someone's finger or elbow, perhaps a jumsie rope or a ball; but generally, the force of impact is rarely as great as when an adult "plays". Some of the more popular sports which are associated with eye trauma include hockey, racket sports and boxing. Of course, periorbital and ocular impact incurred during brawls, street fights and motor vehicle accidents also account for many presentations.

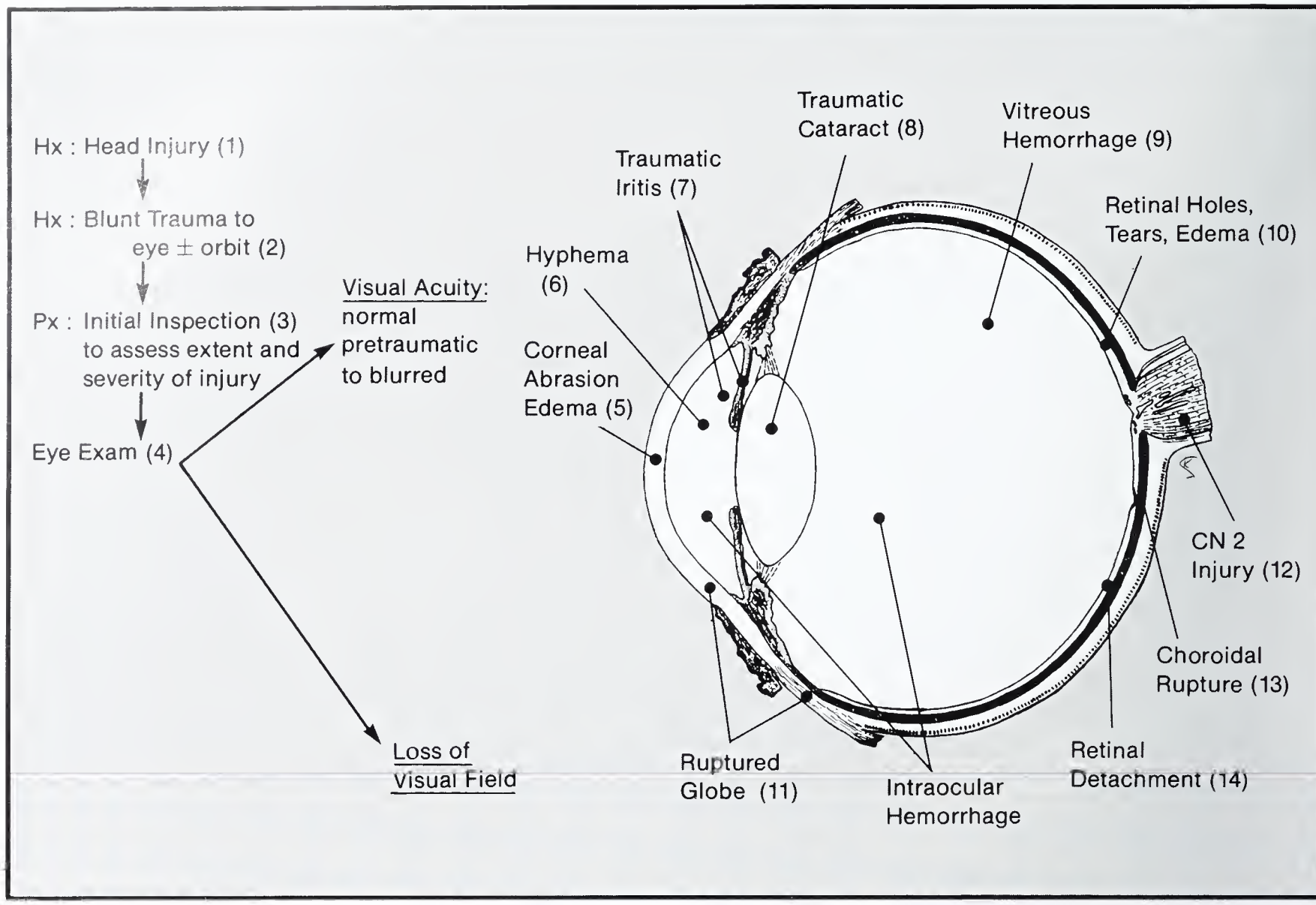
The immediate priorities of the situation focus on CNS injury, assessing both head and spinal trauma, then the possibility of loss of vision. The eye exam is initiated in the perioribital region. Any deformities (hematomas, fractures) are evaluated only as indications of the force of impact as well as the dispersion or concentration of that force. Palpebrae may be involved with hematomas or even lacerations involving the punctae draining to the lacrimal duct. Treatment of these structures must await ocular assessment.

The exam proceeds from the conjunctive through the anterior and posterior segments and is completed with a funduscopic examination. Findings along the way should never satisfy the physician until the integrity of the visual system is assured; otherwise, a retinal detachment underlying a vitreous hemorrhage may go unnoticed. Furthermore, the presence of penetrated matter should be ruled out.

Some of the injuries may not become detectable for days or even weeks following the insult. Follow-up of intraocular pressure is therefore critical, especially if damage to the iris root is suspected. The patient should also be counselled regarding retinal detachment since trauma may predispose one to detachment up to years afterwards.

The purpose of the algorithm presented here, is not to completely analyse the decision-making process to the yes/no level that a computer does. Instead, one might be better served by providing a brief, but digestible perspective of problem-solving. Multiple concurrent diagnoses, complications, and debatable treatments are some very important aspects not dealt with in a decision tree. However, a brief presentation of the problem, as discussed in the following 14 points, should enable the student to 1) quicken the assimilation of information for clinically-oriented problem-solving; and 2) expand the student's problem-solving repertoire.

1. All blunt ocular trauma should be recognized first as Blunt Head Trauma. Although most facial bones act to absorb impact and protect the brain [1], the possibility of closed head injury is kept in mind throughout the investigation. Ominous ocular signs include pupillary dilatation, papilledema, and extraocular muscle paralysis [1,2].


Hx : Head Injury (1)
Hx : Blunt Trauma to eye ± orbit (2)
Px : Initial Inspection (3) to assess extent and severity of injury
Eye Exam (4)
Visual Acuity: normal pretraumatic to blurred
Loss of Visual Field
Traumatic Cataract (8)
Vitreous Hemorrhage (9)
Traumatic Iritis (7)
Retinal Holes, Tears, Edema (10)
Hyphema (6)
Corneal Abrasion Edema (5)
CN 2 Injury (12)
Choroidal Rupture (13)
Retinal Detachment (14)
Ruptured Globe (11)
Intraocular Hemorrhage

Cerebral blindness caused by a coup-contrecoup mechanism is rare. The prognosis is good with maximal recovery in one to 19 weeks [1,2]. A CT scan is indicated to exclude active hemorrhage.

2. The object inciting the Blunt Eye Trauma is often associated with a characteristic injury [3,4]. For example, a pure blowout fracture may be suffered from squashball impact, the eye being decompressed through a periorbital sinus. In contrast, an orbital fracture from dashboard impact will likely occur directly. Also, the friability of the impinging material may increase suspicion of foreign matter within the eye.

3. Some of the more evident signs of Ocular/orbital Trauma are: lacerations, periorbital edema, chemosis, bony defect, enophthalmos, extraocular muscle defect, traumatic iritis, subconjunctival hemorrhage, corneal abrasion, and edema[3,4].

4. The Eye Exam takes priority in spite of disfiguring lid or orbital trauma. Examination under general anaesthetic is indicated if the investigation would otherwise be impaired [5], for example by altered anatomy (edema, fracture) or an uncooperatrive patient (pain, fear). Examination of the untraumatized eye is also essential, watching for the possible development of a sympathetic iritis.

5. Corneal Abrasion is characterized by the following: acute pain on blinking, increased tearing, photophobia, foreign body sensation. Topical anaesthetic may be necessary for a complete eye exam before doing an investigation with fluoroscein dye[6]. Under illumination with a cobalt blue lamp, look for a positive Seidel's test showing aqueous leakage. Vertical scratch marks call for close examination of the upper lid's conjunctival surface looking for a foreign body. Topical anaesthetic is used only to facilitate diagnosis since it delays re-epithelialization and masks complications[6,7].

Tx: Neosporin ointment with a semipressure patch. Disciform Edema, appearing as sharply delineated corneal haziness, results from stromal edema and folds in Descemet's membrane. As with a subconjunctival hemorrhage, it usually clears spontaneously, though a tear in the membrane may be complicated by cicatrization[8].

6. Hyphema. A hemorrhage into the interior chamber may appear as a red tinge in an early presentation. Within hours of the trauma, a red fluid level will evolve. The eye exam must nevertheless be completed. Hyphemas are often associated with drowsiness of unknown mechanism[2,9], but a closed head injury must first be ruled out. The most probable time for a recurrent hemorrhage is within five days after the initial insult. Evidence of corneal staining is an indication for surgical evacuation[10]. Fibrin and clot blocking the trabecular meshwork or pupil may also complicate the injury with the development of glaucoma[6,11].

Tx: bilateral patching, sedation, non-aspirin analgesics (tylenol with codeine), bed rest with 30-40° head elevation to minimize central corneal staining (aminocaproic acid may be used to stabilize the clot).

7. Contusion of the iris can cause either a transient mydriasis from paralysis of the sphincter or a spastic

miosis. Similarly, contusion of the ciliary muscle may result in a short lived hypermetropia or spastic myopia[12]. Traumatic Iritis is characterized by the following: miosis, ciliary injection, anterior chamber flare and cells (slit lamp), blurred vision and photophobia. It is important to examine both eyes for the development of sympathetic iritis.

Tx[13]: cycloplegic mydriatic (scopolamine 0.25%, 1 drop t.i.d.), topical steroid (1 drop every 1 or 2 h until taxonomic response is observed). Sunglasses as desired.

An iris tear is treated identically. A permanent pupillary deformity will result, but it generally does not cause significant visual impairment.

8. Examination of the lens for Traumatic Cataract may be performed with either a slit lamp or an ophthalmoscope (P= +12). In the case of subluxation one might visualize through the slit lamp an anterior chamber of abnormal depth with or without vitreous humour. Iridodonesis is apparent and vision may be unstable, distorted or diplopic.

Tx: Acutely, post trauma glaucoma, whatever the cause, is treated with: miotic agents (pilocarpine) and/or CAH inhibitor (acetazolamide 500 mg iv) and/or mannitol 20% 2g/kg iv.

If vision is unaffected, laser iridectomy may suffice for phacolytic glaucoma. Otherwise, surgery is indicated.

9. Vitreous Hemorrhage presentations run the gamut from asymptomatic to sudden loss of vision. If the retina cannot be visualized, ultrasound is indicated, since a retinal tear with detachment may be the cause of the hemorrhage. If, over the next three to six months, the hemorrhage does not clear spontaneously, a vitrectomy may be required. A rare complication from the organization of the hemorrhage is retinal detachment[15].

10. Macular Holes, although symptomatic, rarely lead to retinal detachment. In contrast, a peripheral tear does predispose to detachment, but is often asymptomatic. The proposed mechanism involves a shearing force generated between the vitreous and the wall[16]. Light flashes from retinal damage and floaters from altered vitreous humour are associated symptoms. Treatment of peripheral tears is with laser.

Retinal Edema is similarly evaluated by funduscopy. In the macular area, blurred vision may progress to profound irreversible central blindness as a hole develops. Peripheral edema and holes may be asymptomatic. The efficacy of osmotic agents is uncertain since the pathology seems related to a reflex vasospasm causing anoxia[17,15].

11. In severe blunt ocular trauma, a Ruptured Globe may result from decompression through the thinnest part of the wall, commonly beneath the recti or the superonasal limbus[18]. Signs and symptoms are: hypotonic eye, deep anterior chamber, intraocular hemorrhage, loss of vision and pain.

Tx: protective shield, admit for surgery.

12. Optic Nerve Injury may be accompanied by blindness or weak pupillary response. Funduscopic findings are a pale, edematous disc with splinter hemorrhages.

Tx: osmotic agents (mannitol 20% 2 g/kg iv). If a CT scan shows canal compromise, emergency decompression is required[19].

13. Trauma to the posterior segment of the eye may cause Choroidal Rupture, which is characterized by an increasing central scotoma. On funduscopy, a white or pigmented ring, concentric with optic disc is visualized.

Tx: initial bed rest and follow-up for possible chorioretinitis[20]. There is no effective treatment.

14. Retinal Detachment is readily diagnosed by loss of visual field and funduscopic examination in which one sees a grey veil of retina with irregular texture. Odds are that a rhegmatogenous (tear-induced) detachment will continue to spread without surgery [15,17]. This is in contrast to a spontaneous detachment which partially resolves with bed rest for 2-3 days before surgery is required.

Acknowledgements

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Prosthetic Cardiac Valves in Toronto

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Abstract

Cardiac valve replacement is often necessary in patients with valvular lesions not amenable to drug therapy or repair techniques. Currently in Toronto, five valve types, classified into two major categories, are commonly implanted. The Bjork-Shiley Monostrut and the St. Jude Medical prostheses are mechanical valves; the Carpentier-Edwards, Hancock II and Medtronic Intact bioprostheses are porcine tissue valves. The Hancock and Ionescu-Shiley Bovine Pericardial prostheses have been discontinued, but are still found in a large proportion of valvular heart disease patients who have had surgery in Toronto. The major complications which affect patients with prosthetic valves are thromboembolic events, endocarditis and mechanical failure or early degeneration of the valve itself. Hemorrhage related to anticoagulant therapy is of concern particularly in mechanical valve recipients as they require life-long therapy. However, mechanical valves have proven more durable than the bioprosthetic valves. In terms of hemodynamics, mechanical protheses generally have a lower mean gradient than the porcine bioprostheses.

Introduction

Valvular heart disease continues to be a problem leading to debilitating disease and premature mortality. This paper is a review of the prosthetic cardiac valves currently found in the adult population who have had surgery performed in Toronto. The availability of cardiac valves has allowed for alleviation of symptoms caused by stenosis and regurgitation in patients suffering from valvular heart diseas.

In general, adult patients receiving prosthetic valves suffer from valve degeneration as a result of valvular sclerosis, rheumatic heart disease, endocarditis, myxomatous degeneration, ischaemic and congenital heart disease.[1] The frequency with which each lesion occurs varies according to population. The results of these lesions are valves which may be stenotic, regurgitant or both. The valves most commonly affected in adults are the mitral and aortic. The tricuspid may become affected primarily by the disease process (e.g. rheumatic fever, carcinoid disease) or secondarily because of right-sided pressure overload as a result of left-sided heart disease.

All forms of valvular heart disease can cause symptoms which are related either to volume or pressure load on the myocardium. Significant valvular heart disease and myocardial dysfunction can be present without disabling symptomatology. Eventually, hemodynamically significant valve lesions can lead to overt failure of the heart as a pump. Certain symptoms are more common in some lesions than others, although at end stage dyspnea and fatigue are common in all lesions. For example, those with aortic stenosis may present with angina pectoris and syncope as a result of ventricular hypertrophy and decreased perfusion of the coronary and carotid vessels.

Prosthetic valves are categorized into mechanical (totally synthetic) and bioprosthetic (tissue cups and synthetic supporting material). The three components of the prosthetic valve include the occluder, the skeleton and the sewing ring. The occluder in the mechanical valve is the poppet whereas in the bioprosthetic valve, it is composed of the tissue cusps. The skeleton in some mechanical valves serves as a cage to keep the occluder oriented; in other mechanical valves and all bioprosthetic valves, the skeleton serves as an attachment point of the occluder. In bioprostheses, the skeleton is known as the stent. The sewing ring, which is supported by the skeleton, provides material for suturing (See figure 1).

HISTORY

The Mechanical Valves

In 1952, Hufnagel implanted the first caged-ball prosthesis in the descending aorta to improve aortic insufficiency before the advent of cardiopulmonary bypass surgery.[2] The next major advancement was the double-caged Starr-Edwards valve (first implanted in 1960) which used a silastic poppet. This valve was changed to a single cage with a silicone rubber poppet and Teflon® sewing ring.[2,3] In 1965, the tilting disk (Kay-Shiley valve) was introduced with this alternate form of

occluder. The Bjork-Shiley Monostrut (tilting disk) and St. Jude Medical (bi-leaflet valve) are currently the mechanical prostheses implanted in Toronto.

The major complications of the early mechanical valves were thromboembolism, hemolysis and early structural failure. Hemolysis and structural failure are less of a problem with the newer mechanical prostheses. Thromboembolism persists since blood tends to clot on the metal surfaces and life-long anticoagulant therapy is necessary in these patients, the main complication of which is hemorrhage.

The Bioprosthetic Valves

Bioprosthetic valves were developed as a result of thromboembolic complications encountered with the mechanical valves. Recipients of bioprosthetic valves are less likely to require life-long anticoagulant therapy as a result of this decreased incidence of thromboembolism. Hence, hemorrhage related to anticoagulant therapy can be avoided, provided that these patients are not in need of therapy for enlarged left atrium, atrial clot or chronic atrial fibrillation. However, these valves have proven less durable than the mechanical valves.[3] Fresh cadaveric human aortic homografts were implanted by Ross in 1962.[4] Autografts from the patient's own fascia lata were implanted by Bjork.[2] Pericardial autografts were later implanted by Senning.[2] These autografts faced early degeneration primarily related to preparation and preservation techniques. The Hancock I porcine xenograft was the first commercially-available bioprosthetic valve.[5] Glutaraldehyde fixation of bioprosthetic valves was found to enhance longevity and durability by increasing collagen crosslinking and decreasing antigenicity.[5] As well as undergoing glutaraldehyde fixation, all currently-used bioprostheses are treated with surfactant to decrease tissue mineralization. The fixation process of the Hancock II and Medtronic Inctact prostheses is carried out at pressures much less than physiological pressure in an attempt to increase mechanical durability. Johnson and Johnson Cardiovascular, the manufacturer of the Hancock II bioprosthesis, claims that low pressure fixation at 1.5 mmHg as opposed to physiological or high pressure fixation allows in maintenance of the collagen crimp and a negligible septal shelf which results in a valve with improved mechanical durability. An optional low pressure fixed Carpentier-Edwards mitral valve is available. The Carpentier-Edwards Porcine, Hancock II Porcine and Medtronics Intact Porcine bioprostheses are currently implanted in Toronto. The Ionescu-Shiley and Hancock Bovine Pericardial valves have been discontinued from clinical use due to concern regarding early valve failure.

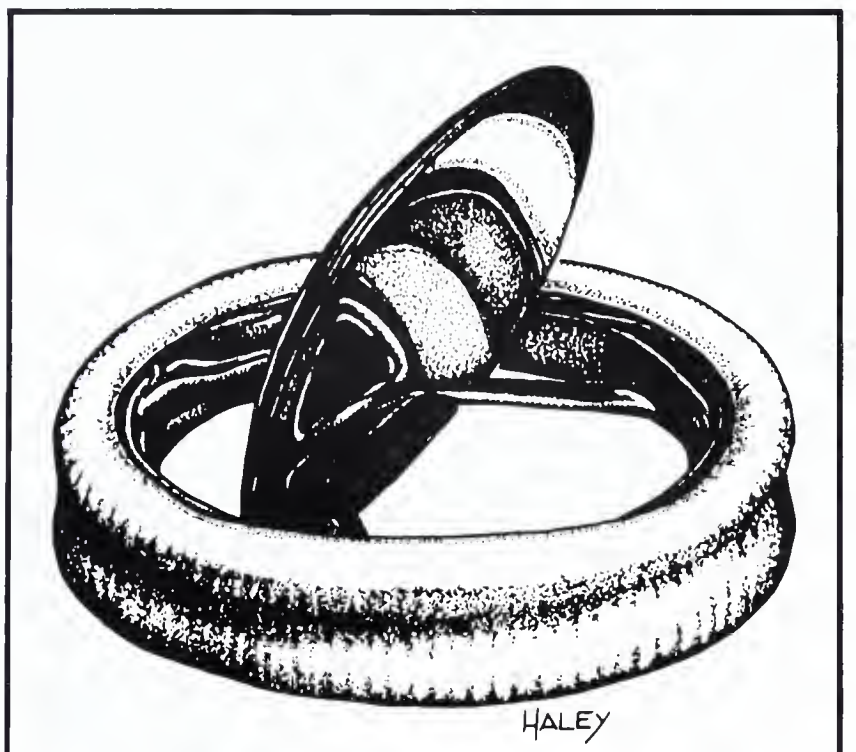

Figure 1 (a): The mechanical valve showing the poppet, cage and sewing ring. In the Bjork-Shiley Monostrut, the poppet is a disc, and cage is the strut.

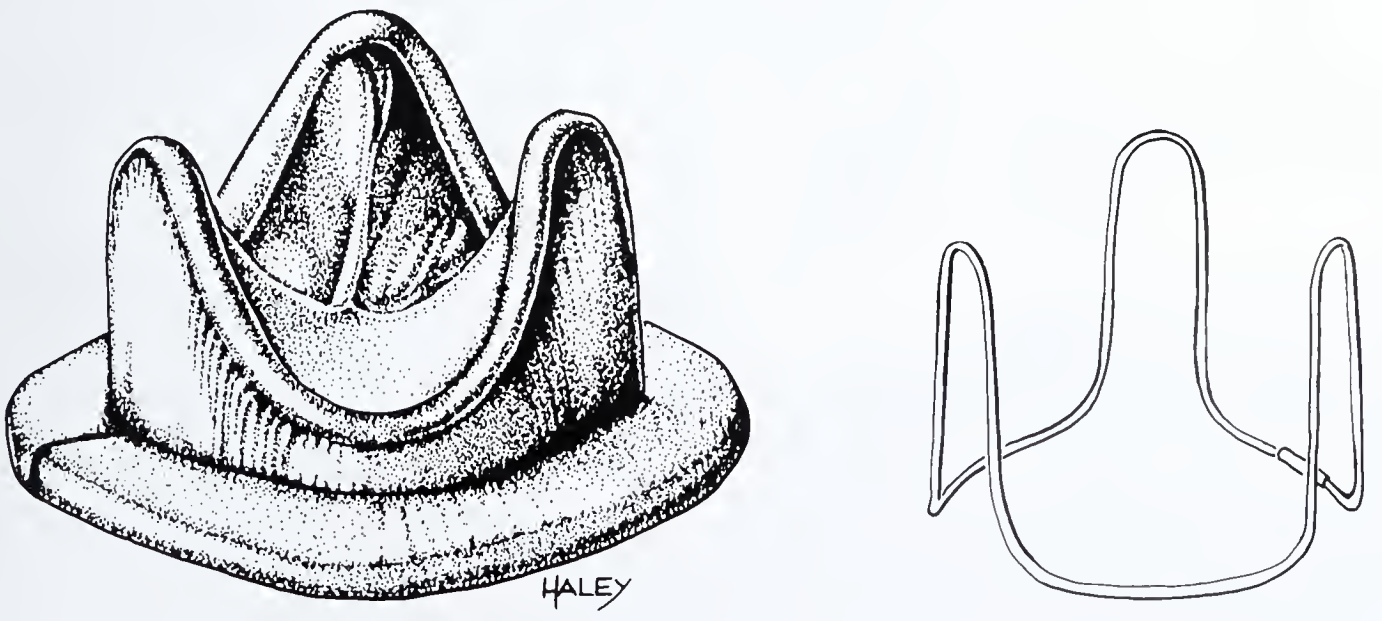

Figure 1 (b): The bioprosthetic valve showing the cusps, stent and sewing ring. The Carpentier-Edwards is shown.

Valve Selection

When selecting a suitable prosthetic valve for replacement, many factors must be taken into consideration. The mechanical valves have proven durability but require life-long anticoagulant therapy and patients may still suffer from thromboembolism despite this therapy. Although not as durable, bioprosthetic valves offer freedom from anticoagulant therapy. This valve type is desirable for those who have an absolute contraindication to anticoagulant therapy, those who plan on bearing children since warfarin crosses the placenta and those who would be non-compliant with taking medication on a regular schedule. Anticoagulant therapy also requires frequent monitoring of prothrombin time which may not be desirable for those who do not have convenient access to facilities providing this. Even though bioprosthetic valves generally do not require anticoagulant therapy, all prostheses have some inherent risk of thromboembolism. Patients may not wish to have a bioprosthetic valve implanted with the knowledge that there is a possibility of undergoing the ordeal again in ten years or so. In such a case, a mechanical valve would be indicated.

The mode of failure of the valve types is also a consideration. Bioprosthetic valves usually show a slowly progressive failure with time for detection and thoughtful consideration of re-operation. Mechanical valve failure is more catastrophic as a result of valve thrombosis, strut fracture or loss of ball or disc from the cage.[6]

Hemodynamic performance in prosthetic valves is also considered in the selection process. The stenosis gradient tends to be lower for the mechanical prostheses as compared to the bioprostheses.[6,7]

THE VALVES

The Mechanical Valves

The Bjork-Shiley Monostrut valve is a low-profile, tilting-disk prosthesis which evolved from the original Bjork-Shiley Delrin® disc valve first emplanted in 1969. The Standard Bjork-Shiley was introduced in 1971 with a pyrolytic carbon disc as this material had less propensity to absorb moisture.[8] The disc was changed in 1976 to a convexo-concave shape which allowed for separation of the valve from the ring in open position to assist in prevention of thrombus formation in low flow areas.[8] The opening angle was increased from 60 to 70 degrees to achieve a ten percent pressure gradient decrease.[8] The 60 and 70 degree convexo-concave valves have been withdrawn from the market as fractures in the outflow strut have been experienced with both valve types,[6,8] The Bjork-Shiley Monostrut prosthesis with 70 degree opening angle is currently implanted. All welds in this valve have been eliminated by manufacturing the entire strut with one piece of alloy[6,8] (Refer to figure 2).

The St. Jude Medical Valve is a low profile, bi-leaflet, tilting disc mechanical prosthesis first introduced in 1977. In contrast to the Bjork-Shiley Monostrut, blood flow is more central. The leaflets open to 85 degrees and close to 30-35 degrees which allows for a high orifice to tissue annulus ratio.[10] This valve is therefore suitable for patients with a small left ventricle or small aortic root.[3]

Bioprosthetic Valves

The Carpentier-Edwards valve, which has been commercially-available since 1976, is composed of glutaraldehyde-preserved porcine tissue cusps on a flexible Elgiloy® stent.[9] The manufacturer claims that the flexibility of the cobalt and nickel stent decreases the stress on the cusps at closure and allows for increased valve durability.[5] The orifice to annulus ratio of the stents was modified in 1980 to improve hemodynamic performance, particularly in the smaller size valves.[5] This change has not been found to make a statistically significant difference in hemodynamics.[5,25] A unique feature of the Carpentier-Edwards valve is the fact that the contour of the stent is assymetric to conform with the natural contour of the porcine valve. As a result, the muscle shelf of the porcine right coronary cusp does not become incorporated into the orifice but is sewn into the stent[11] (See figure 2).

The Hancock II bioprosthesis, first implanted in 1982, differs from the Hancock I in that it has a lower profile, has a stent made of Delrin® rather than polypropylene, and the fixation process is performed under low pressure.[12] The anti-mineralization surfactant treatment of the porcine tissue in the Hancock II valve is called T6® (See figure 2).

The newer Medtronic Intact bioprosthesis has a stent made from radiolucent, injection-molded plastic.[13] This radiolucency makes the valve difficult to visualize radiographically, whereas all other valves discussed are easily visualized as they are radiopaque. A process known as "stress free fixation" in which glutaraldehyde treatment is performed at near zero pressure is utilized during fixation.[14] This also maintains the tissue in relaxed neutral state to retain collagen crimp (See figure 2).

The Hancock Bovine Pericardial bioprosthesis was first implanted in 1982 and later discontinued. The bovine pericardial tissue cusps of gluteraldehyde treated prosthesis were subject to tearing at the site of the stent attachment. On examination of the explanted valves, the collagen bundles were found to be in disarray at the tear sites.[15]

The standard and low-profile models of the Ionescu-Shiley Bovine Pericardial bioprostheses are gluteraldehyde treated bovine pericardial valves with Titanium (standard) or Delrin® (low-profile) stents and Dacron® sewing rings.[9] These valves have been discontinued since first implantation of the standard model in 1976. Towbridge et al reported commissural tears in association with commissural sutures in valves explanted due to regurgitation.[16]

Complications

Patients with valvular heart disease are prone to certain complications, some of which are related to the disease (e.g. endocarditis), some to the therapy (e.g. anticoagulant-related hemorrhage), and some to the prosthesis (e.g. thromboembolism, thrombus). Rates of thrombus formation for mechanical and tissue valves are negligible. Table 1 shows a comparison of linearalized rates of endocarditis and prosthetic malfunction as well as actuarial rates of freedom from death and thromboembolus. Kopf et al[18] and Czer et al[19] reported

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rates of anticoagulant-related hemorrhage of 1.3%/pt.yr. and 2.9%/pt.yr. respectively for the St. Jude Medical valve, whereas Aris et al[23] and Lindblom et al[17] each reported rates of 2.2%/pt.yr. for the Bjork-Shiley Monostrut. In follow-up studies in bioprosthetic valve recipients it was ensured that only patients with a large left atrium, in chronic atrial fibrillation or with an atrial clot were given life-long anticoagulant therapy.[20,21,22] Anticoagulant therapy was only utilized in the study performed by Bortolotti et al in patients receiving a Hancock Bovine Pericardial prosthesis with large left atrium or atrial clot.[15] Therefore, rates of anticoagulant-related hemorrhage for this group are not included. No data were included on the Hancock II or Medtronic Intact prostheses as they are relatively new and long-term follow-up is not yet available.[13]

Hemodynamics

Hemodynamic performance of prosthetic valves is another factor which contributes to the difference between mechanical and bioprosthetic valves. The St. Jude Medical valve has the lowest stenosis gradient in both hemodynamic and hydrodynamic studies.[24,27] Porcine bioprostheses have a higher stenosis gradient. The Bjork-Shiley Monostrut has a gradient somewhat comparable to the porcine bioprostheses. Table 2 shows a comparison of the mean systemic and diastolic gradients of the 25 mm aortic and mitral prostheses.

CONCLUSIONS

Both mechanical and bioprosthetic valves have promising futures and will continue to play an important role in the alleviation of symptoms caused by valvular heart disease. The ideal valve of the future will be one which has the durability of the mechanical valve combined with the freedom from thromboembolism and anticoagulant related hemorrhage of the bioprosthetic valve.

Acknowledgements

We would like to thank Karen Frandsen and Ellie Airey for their assistance in the word processing.

Table 2
Hemodynamic data for aortic and mitral prostheses at rest*.

VALVE POSITION	REF	MEAN GRADIENT (mmHg)
AORTIC		
Mechanical		
Bjork-Shiley Monostrut	23	13 ± 3
St. Jude Medical	24	3.4 ± 1.8
Bioprosthetic		
Carpentier-Edwards**	25	15.9 ± 1.7
Hancock II***	26	7.3 ± 5.5
Hancock Pericardial†	27	4.3
Ionescu-Shiley†	27	6.6
MITRAL		
Mechanical		
Bjork-Shiley Monostrut	23	6.2 ± 2.1
St. Jude Medical	24	1.4 ± 0.2
Bioprosthetic		
Carpentier-Edwards**	25	6.5 ± 0.9
Hancock Pericardial†	27	4.3
Ionescu-Shiley†	27	6.6

* pt. at rest or C.O. < 5.0 l/min
** includes standard and improved annulus models
*** peak systolic gradient
† in vitro hydrodynamic studies

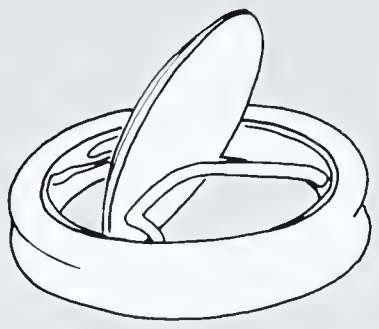
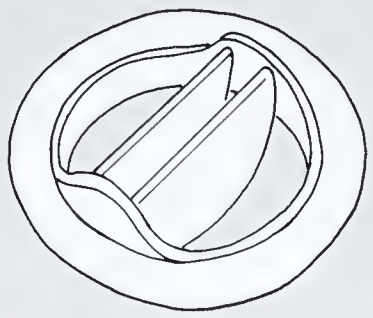
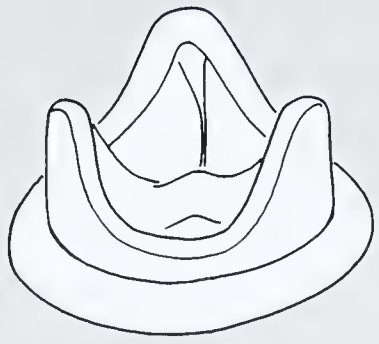
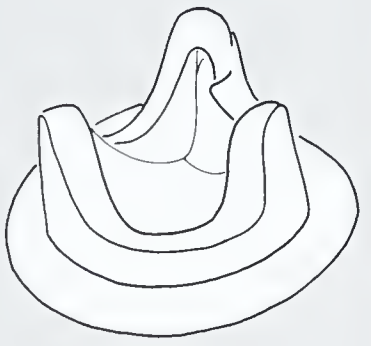
Table 1
Comparison of rates of endocarditis, malfunction and rates of freedom from death and thromboembolism in patients with mechanical and bioprosthetic valves.

Prosthesis	Ref.	Endocarditis	Malfunction	Freedom from Death		Freedom from Thromboembolism	
		%/pt. yr.	%/pt.yr.	post op years	total (5)	post op years	total (%)
MECHANICAL							
Bjork-Shiley	17*	0.4	0	3	88.6		------
	23.	0.2	----	4	94.7	3.5	92.2
St. Jude Medical	10	0.3	----	5**	76	5	89
	18	0.3	0.3***	5**	76.7	5	97.4
	19	0.3	0		-----	8	90
BIOPROSTHETIC							
Carpentier-Edwards†	20	1.3	0.1		-----	5	95.2
	21	0.6	0.1	6**	74.0		-----
Ionescu-Shiley††	22	2.2	1.4	6	78.0	6	95.3
Hancock Bovine Pericardial	15	2.4	3.0		-----		-----

* linearalized data includes single valve replacements only
** includes early (<30 days post-op) death
*** includes thrombus formation
† includes both standard and improved orifice
†† standard model

Figure 2

A comparison of appearance, materials, list price and features of the mechanical and bioprosthetic valves currently implanted in Toronto.

PROSTHESES	BJORK-SHILEY	ST. JUDE	CARPENTIER-EDWARDS	HANCOCK II	INTACT
					
1) **MODEL**	Monostrut	Medical	Mitral 6625 Aortic 2625	Mitral T510 Aortic T505	Mitral 705 Aortic 805
2) **MATERIAL**					
Reference	9	10	9	13	13
Occluder	pyrolytic carbon	pyrolytic carbon	porcine tissue	porcine tissue	porcine tissue
Skeleton	Haynes 25®	pyrolytic carbon	Elgiloy®	Delrin®	injection molded plastic
Sewing Ring	Teflon® or Dacron®	Dacron®	Teflon®	Dacron®	Dacron®
3) **LIST PRICE***	$2,395	$2,500-2,900	$2,240	$2,275	$2,325
4) **FEATURES**	- convexo concave disc - monostrut	- bi-leaflet - central flow	- assymetric - flexible stent - low pressure fixated mitral valve (option)	- low pressure fixation	- stress free fixation - radiolucent

* all list prices were given by the Toronto distributors

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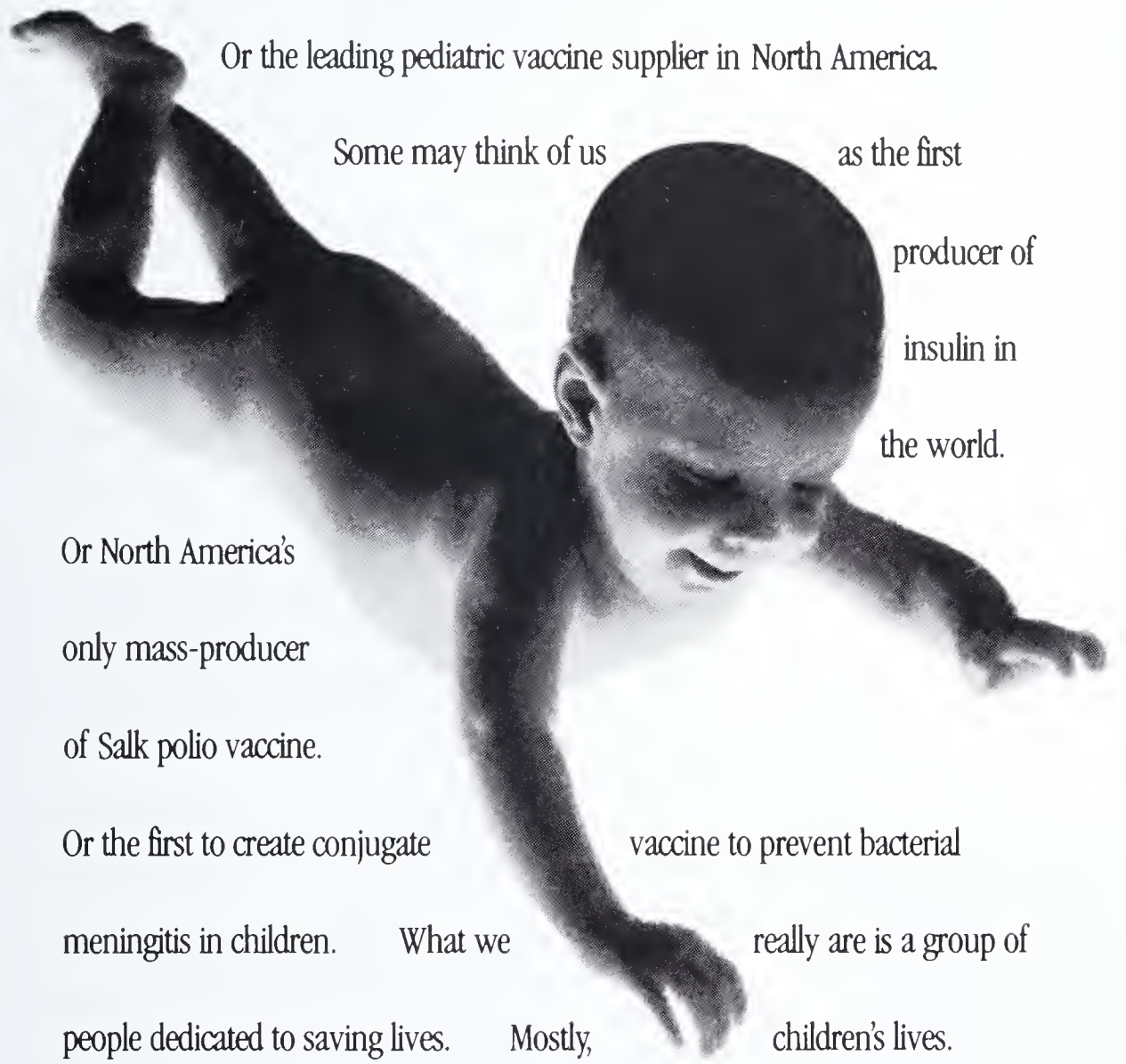

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How to Read a Chest X-Ray: A Systematic Approach

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Abstract

The ability to interpret a chest x-ray (CXR) is essential for all physicians, especially in the earlier stages of their careers. The thought of having to read a CXR can be very intimidating, unless a systematic approach is used. This paper describes such an approach, using extrinsic and intrinsic basics. A mnemonic is suggested to help remember the basic steps. All figures and summaries are displayed on pocketbinder-size pages so that they can be included in the reader's personal binder for convenient reference.

Introduction

The medical profession can be very exciting and rewarding, but it can also lead to frustration and fears of failure. These fears are heightened when the physician is confronted with a task for which he/she is poorly prepared. One area in particular which often elicits such fears is the interpretation of radiological films. The chest x-ray (CXR) is the most common radiological procedure performed. Thus, it is of the utmost importance to know the principles of chest radiograph interpretation. This knowledge is of value not only during medical training, but throughout one's medical career, regardless of specialty. Yet many physicians, due to a lack of an organized approach, find the task of CXR interpretation frustrating.

The intention of this paper is to describe a systematic approach so that the interpretation of the CXR is made simpler. A mnemonic is suggested to assist in recall of the basic steps of interpretation (pocketbinder page 1). To aid in the visualization of the normal anatomy involved, a number of pocketbinder-sized pages (PB) are supplied, which also summarize some of the more common CXR pathology findings.

The approach has two major parts. In the first part, the extrinsic basics, which are the technical factors, are examined. In the second part, the intrinsic basics, which relate to pertinent patient anatomy, are outlined (PB 1 & 2).

The order of basic radiographic densities, namely AIR < FAT < WATER < BONE, is essential information for the interpretation of any radiograph (PB 3). A definition of the 'silhouette sign', which requires an understanding of the radiographic densities, is also supplied (PB 3).

It is important to remember that before any definitive diagnosis is made, all radiographic findings should be correlated with the patient's history and physical examination, when possible.

Part I: The Extrinsic Basics

i) Identification

One should start by locating the patient profile marker on the film. This will supply the patient's name, and the date of the examination. From the name, the patient's sex, and perhaps nationality, can often be deduced. Many centers will also include the patient's age. If the sex cannot be determined from the name, check for the presence of breast shadows. The incidence of many disease entities varies according to the above patient parameters.

ii) Markers

The right (R) and/or left (L) markers should be identified. These may reveal that the film has been put up backwards (a common examiner's trick), or perhaps that the markers have been erroneously placed. Without such consideration, rare conditions such as dextrocardia may apparently become more common!

iii) Position

This refers to the patient's position relative to the direction of the x-ray beam. A 'frontal' CXR refers to either a PA (postero-anterior) or AP (antero-posterior) technique. A PA CXR is taken with the patient's anterior chest wall against the film, with the x-ray source behind the patient, so the x-rays pass from posterior to anterior. The AP CXR is the reverse. The PA technique is more common, and gives a more accurate representation of the heart and mediastinum. To determine whether patient rotation is present on the PA film, compare the medial ends of the clavicles to the midline. The midline is generally determined by a line through the vertebral spinous processes. If the medial end of the left clavicle is closer to the midline, then the patient is rotated to the right, and vice versa.

iv) Quality

The two major factors that determine the quality of the radiograph are the degree of x-ray penetration, and the developing technique. In a good quality CXR, one should just be able to visualize the intervertebral disc spaces through the heart shadow on the frontal film. If the disc spaces are poorly visualized and the film appears too white, then it is underpenetrated or underdeveloped, and vice versa.

HOW TO READ A CHEST X-RAY:

A SYSTEMATIC APPROACH

Part I: The Extrinsic Basics

Identification
Markers
Position
Quality
Respiration

Part II: The Intrinsic Basics

Soft tissues
Abdomen
Bony structures
Central shadow
Hila
Lungs
Absent structures

Mnemonic: Regarding your CXR interpretation:

"It Might Prove Quite Right (but)
Stop And Be Careful How Lungs Appear"

CXR APPROACH DETAILS

EXTRINSICS:

Identification – date of exam, patient name, sex, age, etc.

Markers – R & L

Position – patient rotation eg. medial border clavicles with respect to midline

Quality – degree of penetration, eg. disc spaces visible through heart?

Respiration – R hemidiaphragm at 5th anterior intercoastal space, ~ 10th posterior ICS on PA CXR, on full inspiration

INTRINSICS:

Soft tissue – neck, axillae, breasts, anterior & posterior chest wall

Abdomen – liver, stomach, colon, spleen, vertebrae, aorta

Bones – c-spine, t-spine, shoulder girdle, sternum, ribs

Central shadow – trachea, heart borders, great vessels

Hila – vessels, bronchi, nodes

Lungs – pleura, diaphragm, lung parenchyma

Absent structures – review intrinsic basics & look

USEFUL X-RAY MISCELLANEOUS

a) **Basic Radiographic Densities with X-Ray Appearance:**

Air < Fat < Water < Bone

Black <————————> White

b) **Cardio-Thoracic (CT) Ratio:**

$$\frac{\text{Max. Transverse Diameter Heart}}{\text{Max. Transverse Diameter Chest}}$$

CT Ratio > 50% is abnormal, indicating likely cardiomegaly (on PA CXR with full inspiration)

c) **'Silhouette Sign':**

A structure will be visualized if its radiographic density is different from that of the adjacent tissue.
With adequate penetration, a structure that is not visualized, but should be, indicates pathology.

STRUCTURES COMPRISING THE CARDIAC SHADOW ON CXR:

a) **Frontal Film:**

b) **Lateral Film:**

v) **Respiration**

The CXR is taken at maximal inspiration to avoid blurring during respiratory movements, to expand the lungs fully to enable easier visualization of the pulmonary bases, to allow for a more accurate estimation of the cardio-thoracic (CT) ratio (PB 3), and to allow for reproducibility of that position in subsequent CXRs of the same patient.

Part II: The Intrinsic Basics

One of the most common errors made during CXR interpretation is to concentrate solely on suspected pulmonary or cardiac pathology. Significant information may be gained when the soft tissues, bony structures and abdomen are examined. Abnormalities in these areas may give the diagnosis, or point toward specific heart or lung pathology.

Be sure to apply the approach to the lateral CXR, as well as the PA film. Apart from providing detailed information regarding the mediastinum and lung bases, the lateral film may also permit localization of a lesion identified on the frontal film. The frontal film allows for right/left differentiation, and the lateral film will determine in which lobe a pulmonary lesion is located!

i) **Soft tissues**

When examining the soft tissues (STs), start from the neck and work down. The routine should include the neck, both axillae, the pectoral muscles, the breasts and nipples, and the diaphragm. The diaphragm, due to its intimate relationship with the lungs, will be discussed in the section on pulmonary interpretation.

The normal ST findings which can simulate pulmonary pathology include the apparent increase in lung density in the lower lobes due to overlying breast tissue (check the lateral film); the appearance of a pulmonary nodule due to a nipple shadow (review the lateral projection, or repeat the frontal film using metallic nipple markers); and on the lateral film, the illusion of increased lung density in the apices, due to the overlying ST and bone densities of the upper extremities (review the frontal film).

ii) **Abdomen**

Depending on the film technique and the size of the patient, one may be able to view up to half the abdominal contents. The normal structures to recognize on the frontal projection include the liver shadow under the right hemidiaphragm, the gastric bubble medially under the left hemidiaphragm, the spleen just lateral to the stomach, and occasional loops of air-filled bowel. On the lateral film, take note of the vertebral column, and, if calcified, the abdominal aorta.

Some abnormalites to check for include: organomegaly, calcification within viscera, free air in the retroperitoneum, peritoneum or subcutaneous fascial planes, surgical clips, or radiological contrast material. Some of these findings, along with fractures and degenerative vertebral changes, should also be noted with respect to bony structures (see below).

iii) **Bony structures**

The routine involving the skeletal structures should proceed from the cervical and thoracic vertebrae, to the shoulder girdle (clavicles, scapulae, proximal humeri), the ribs, costal cartilage and the sternum.

When inspecting the vertebral column, note the alignment, the vertebral body height and appearance, disc space height, pedicle pairs per vertebra, and the spinous processes. To view the ribs optimally, turn the CXR on its side - this will allow concentration on the rib anatomy without interference from the central shadow or lungs. The lateral projection is preferred for examination of the sternum.

iv) **Central shadow**

The major components of this region are the trachea and carina, the heart shadow, and the superior, anterior and posterior mediastinal areas.

The trachea should be followed along its entire course from the neck to the carina. It should lie in the midline except for a slight deviation to the right just above the carina, due to displacement by the aortic arch. Tracheal deviation without significant patient rotation suggests the presence of pathology (i.e. displaced to the opposite side with tension pneumothorax or extrinsic mass, or to the same side with volume loss or disease-related pulmonary cicatrization).

Knowledge of the normal cardiac appearance enables one to identify isolated enlargements, or other abnormalities, of the various chambers and vessels (PB 4). On the frontal film, the right heart shadow border is made up by the superior vena cava, the right atrium, and the inferior vena cava. The left cardiac border is made up by the left subclavian vessels, aortic knuckle (aortic arch), pulmonary artery, left atrial appendage and the left ventricle, surrounded by a variable amount of pericardial fat. The right ventricle is not normally border forming on the PA CXR radiograph. Valvular calcifications, typically aortic or mitral, are not uncommon in the elderly population, and are best seen on the lateral CXR (PB 5).

The anterior mediastinum is the space anterior to the pericardium and trachea, including the retrosternal air space. Differential diagnosis (DDx) of a mass in this region includes the 'four T's' (thyroid, thymus, teratoma, thoracic aortic aneurysm), as well as lymphoma and pericardial cysts. The middle mediastinum contains the heart and great vessels. A mass in this area might include lymphadenopathy, carcinoma of the bronchus and multiple cardiovascular causes. The posterior mediastinum is behind the posterior pericardial surface. The DDx of a mass here would include GI hernias, aortic aneurysm and neurogenic tumors (a neurolemoma), among others.

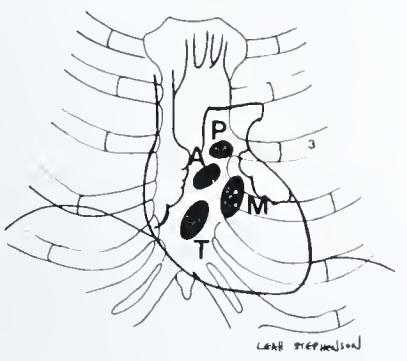
v) **Hila**

The hilum of the lung contains the mainstem and primary segmental bronchi, the pulmonary vessels, and lymph nodes. The left hilum is slightly higher than the right in the normal individual. The pulmonary vessels contribute most to the hilar density, with the normal lymph nodes being too small to be identified. The pulmonary veins can often be distinguished from the arteries, since the veins lie below, or lateral to, the arteries,

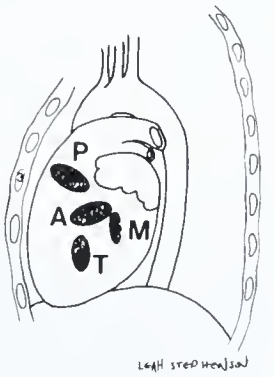
5

LOCATIONS OF CARDIAC VALVULAR CALCIFICATIONS ON CXR:

a) **Frontal Film:**

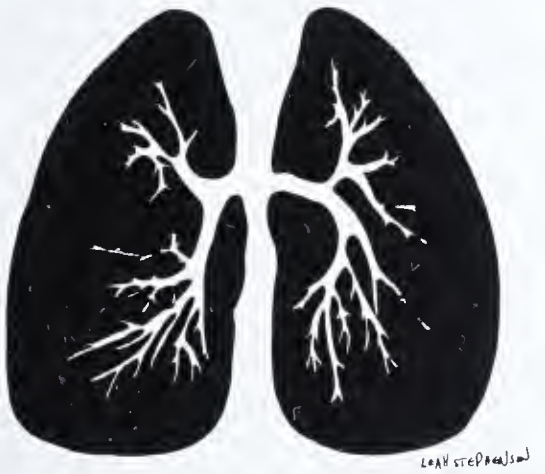


b) **Lateral Film:**



NORMAL PULMONARY VASCULAR PATTERN ON PA CXR:

Lower lobe vessels > upper lobe vessels on upright PA CXR



6

BASIC PULMONARY PATHOLOGY:

I) AIRSPACE VS. INTERSTITIAL LUNG DISEASE

Airspace Disease:
- small (5-10 mm), fluffy, ill-defined coalescent nodules
- air brochogram/air alveologram
- may spread rapidly, via pores of Kohn

Interstitial Disease:
- Nodular type: tiny (2-3 mm) multiple, discrete nodules
- Reticular type: thin (1-2 mm) well defined linear densities, often network-like
- Reticular-Nodular type: a combination of the above

II) SIGNS OF MALIGNANCY IN A PULMONARY NODULE:

	Malignant	**Benign**
Margin:	ill-defined	well defined
Contour:	lobulated	smooth
Calcification:	none or eccentric	often present
Growth Rate: (doubling time)	10-465 days	< 10 or > 465 days
Associated Signs:	lymphadenopathy, bone destruction, pleural effusion	often none

7

SUMMARY OF RADIOLOGIC FINDINGS ASSOCIATED WITH COMMON DISEASE PROCESSES SEEN ON CXR:

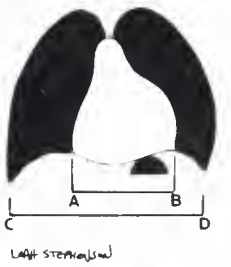
i) **CARDIOVASCULAR:**
- a) **congestive heart failure** (see PB8) - LV enlargement, vascular redistribution, pulmonary edema, Kerley's B lines
- b) **pulmonary hypertension** - RA & RV enlargement, prominent central pulmonary vessels at hila, rapid tapering of vessels, avascular peripheral lung zones
- c) **systemic hypertension** - LV hypertrophy, prominent aorta
- d) **mitral stenosis** - LA enlargement, prominent pulmonary veins, mitral calcification, Kerley's B lines
- e) **aortic stenosis** - calcified aortic valve, LV enlargement, prominent aorta

ii) **PULMONARY:**
- a) **interstitial vs. airspace** (see PB 6)
- b) **viral pneumonia** - nodular, diffuse infiltrate
- c) **pneumococcal pneumonia** - lobar infiltrate and consolidation, with air bronchograms, ± pleural effusion
- d) **tuberculosis** - apical infiltrate, parenchymal Ca^{++}
- e) **granulomas** (TB, histoplasmosis, etc.) - fibrosis, Ca^{++} nodules
- f) **emphysema** - overexpanded lungs, low diaphragm, increased radiolucency of lungs, apparent small heart
- g) **pneumothorax** - visceral pleural line visible, extrapulmonary intrathoracic air

8

COMMON PA CXR FINDINGS OF CONGESTIVE HEART FAILURE:

a) **Cardiomegaly** - CT ratio > 50% LV enlargement typically



b) **Vascular redistribution** - upper lobe vessels > lower lobe vessels in upright PA CXR film

c) **Kerley's B lines** - fluid in interlobular septa, at lung bases, seen as thin white lines extending to the lateral chest wall

d) **Pulmonary edema** - interstitial fluid surrounds vessels to produce a hazy vascular pattern, as well as patchy airspace infiltrates

and empty into the left atrium at the lower margin of the hila.

It is difficult to distinguish the cause of mild hilar enlargement on a plain CXR, and other radiological modalities are usually required for clarification. In terms of forming a differential diagnosis, it is important to discern whether hilar enlargement is unilateral (likely carcinoma of the bronchus, or infectious etiology) or bilateral (lymphoma or sarcoid). Pulmonary artery enlargement may be unilateral or bilateral.

vi) **Lungs**

The diaphragm, the pleura and the lungs proper will be discussed separately.

a) Diaphragm: The relative position of the diaphragmatic silhouette on a CXR depends on many variables, including patient position, respiration, and lung volumes. On full inspiration, the right hemidiaphragm is generally at the level of the fifth anterior interspace, while the left dome is usually slightly lower. One is usually able to distinguish the right and left domes on the lateral film: the left dome is obscured anteriorly by the heart, the 'gastric bubble' is directly below the midsection of the left dome, and the left major pleural fissure is more vertical inferiorly and intersects the left dome more posteriorly than occurs on the right.

The acute angle formed where the diaphragm meets the chest wall is the costophrenic angle. It is normally sharply defined, but it may be blunted or obliterated in association with diseases causing pleural effusions, adhesions or thickening.

b) Pleura: When inspecting the pleura, follow the entire perimeter of the bony thorax and look over both domes of the diaphragm. On occasion, one can identify the minor fissure on the frontal CXR, and the minor and major fissures on the lateral film. The pleura is visible in certain pathological states, such as pneumothorax (the pleura appears as a thin line between the lung and the extrapulmonary intrathoracic air), pleural effusions, and pleural thickening (check the apices - eg. post granulomatous disease). If a mass is pleural or extrapleural in origin, it will form obtuse angles at its edges, in comparison to the acute angles of pulmonary pathology abutting the pleura.

c) Lungs: To interpret the lungs, make careful comparison of the relative symmetry of each paired intercostal space, using the posterior rib outlines as guides. Students and younger physicians with less experience may find it helpful to hold a piece of paper against the film and slowly lower it to assist side to side analysis. It is imperative that the examination of the lungs continues behind the cardiac shadow and below the upper silhouette of the diaphragm on the frontal film (use the lateral view for comparison).

The x-ray appearance of the lungs is due to the air-filled alveoli (black) and the contrasting pulmonary vessels (white). The larger airways are difficult to distinguish on the normal CXR. When the patient is upright, the upper lobe vessels are smaller than those at the bases, reflecting the gravity dependent blood flow distribution (PB 5). 'Vascular redistribution' refers to abnormal prominence of the upper lobe vessels on an upright CXR, and suggests underlying pathology, as with congestive heart failure (PB 7 & 8).

Although the text of this paper has been designed to discuss the normal radiologic anatomy, some commonly encountered examples of CXR pathology are included with the pocketbinder pages. A point form discussion of interstitial and airspace disease, and the characteristics of benign vs. malignant pulmonary nodules are included on PB 6. A summary of x-ray findings associated with other common CXR disease processes is outlined on PB 7, and the classical PA CXR findings of acute congestive heart failure are diagrammatically displayed on PB 8.

vii) **Absent structures**

It is important to be alert for the absence of structures. With this in mind, one should quickly review the examination of the intrinsic basics. Common oversights include the absence of one or more ribs, a breast shadow, or perhaps the lobe of a lung. Surgical removal is the most common cause of absent structures, but occasionally the abnormality is congenital.

Summary

As a medical practitioner, the ability to interpret a CXR is vital. There is little exposure to radiographic interpretation techniques during medical school, and graduating doctors often have insufficient knowledge of how to discern normal anatomy from significant pathology. The solution is to have a systematic interpretation approach.

This paper has proposed such an approach by combining the evaluation of technical factors, termed the **Extrinsic Basics**, and the essential steps of anatomical scrutiny, designated the **Intrinsic Basics**. These are as follows: extrinsics - Identification, Markers, Position, Quality, Respiration; intrinsics - Soft tissues, Abdomen, Bony structures, Central shadow, Hila, Lungs, Absent structures. A **mnemonic** was devised to aid recall of these steps in order: Regarding one's CXR interpretation, **"It Might Prove Quite Right** (but) **Stop And Be Careful How Lungs Appear".**

The multiple pocket-binder mini pages provided will allow quick, portable reference to the important summarized points and diagrams necessary for correct CXR analysis.

Acknowledgements

Dr. L. Fraser and Dr. E. Becker, of the Toronto Hospital, Western and General Divisions.

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Researching the Future

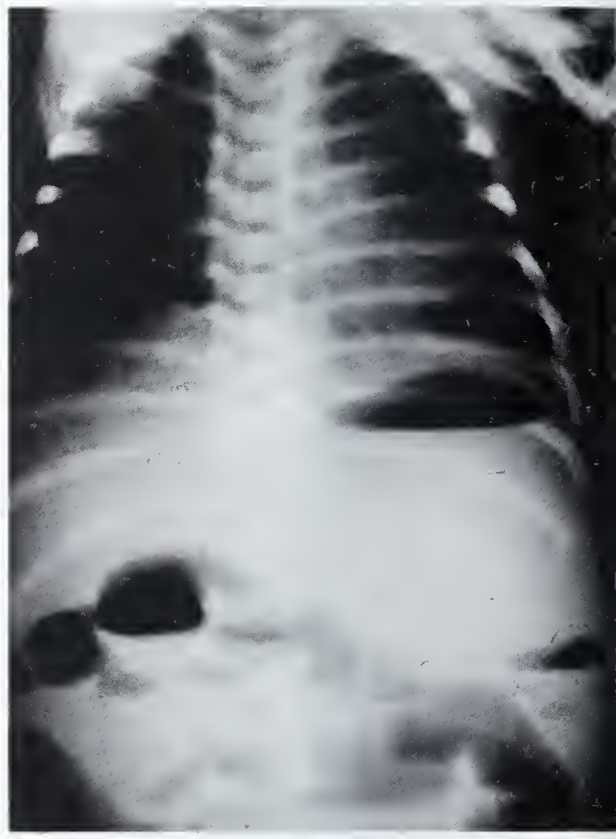
Quick Dx-1

R.M. Filler and Michael J. Weinberg*

**Surgeon-in-Chief, The Hospital for Sick Children*

Case 1

Case 2



Case 3

Case 2

Questions

1. What is the cause of intestinal obstruction in each of these newborns?

2. What are the common presenting features?

3. What is the treatment of choice?

Answers on page 29.

Case 3

Quick Dx-2

Michael K. McLennan, B.Sc., M.D. (8T6)

A

B

C

D

These four radiographs are associated with four different clinical histories. Match the x-ray to the history.

1) Middle aged patient with sudden onset of unilateral chest pain and marked shortness of breath, following a failed central line insertion attempt.

2) Middle aged patient with no acute symptoms, for general surgery clinic follow-up.

3) Recent laparotomy, now has pleuritic chest pain, purulent sputum, and shoulder tip pain.

4) Young patient, asymptomatic, required CXR for commencement of employment.

Answers on page 29.

Iridiocorneal Endothelial Syndrome: Its Causes and Effects

Catherine Birt, M.A., 8T9

Abstract

Iridiocorneal endothelial syndrome is a disease of the endothelium of the cornea of the eye. It has three subtypes which are differentiated on the basis of gross clinical appearance, but the subtypes are considered to result from a single pathogenetic cause. This paper discusses the appearance of the different forms of the ICE Syndrome -Progressive Iris Atrophy, Chandler's Syndrome and Cogan-Reese Syndrome - and current theories of their pathogenesis.

Introduction

Iridiocorneal endothelial syndrome (ICE Syndrome) is a rare condition producing unilateral glaucoma with characteristic iris changes.[1,2] It affects the anterior chamber of the eye, specifically the iris and the corneal endothelium - hence the acronymic name of the syndrome.[3]

The first part of this paper describes the three subtypes of the ICE Syndrome, their typical presentations, clinical course and management. The second section discusses current theories about the etiology of the condition.

Clinical Presentation

The ICE Syndrome consists of three conditions which had previously been differentiated on the basis of their clinical appearance. These conditions - progressive iris atrophy, Chandler's Syndrome and Cogan-Reese or Iris Nevus Syndrome - are now considered to be a spectrum with one basic pathogenesis,[3] since although pure forms of all three are easy to find, cases with overlapping features are also seen.[6,7]

Progressive iris atrophy (PIA) was first described in a German paper by Harms in 1903[8] and was first defined in 1965[9] as having iris changes as the principle feature, with variable amounts of corneal edema, endothelial dystrophy and the formation of peripheral anterior synechiae (PAS) also seen. Loss of stroma and pigment epithelium results in large, irregular holes in the iris. Other iris changes include ectropion uveae (the dragging of the normally posterior pigment epithelium to an anterior position), corectopia (an irregularly shaped pupil), and displacement of the iris collarette (the fringe of iris around the pupil).[2,4,8] Corneal changes include stromal edema and an endothelial appearance often described as resembling "fine beaten silver"[8] or "hammered silver".[10] Most patients have features of both iris and corneal changes.[8] The other major feature in PIA is the presence of glaucoma within the affected eye only.[2,3,11] These changes are found with equal frequency in both left and right eyes, but within one patient the contralateral eye is usually not affected.[7]

Chandler's Syndrome, first described by Chandler in 1956[12] may also have features of iris atrophy and glaucoma, but these are late and mild.[13] The most important changes are endothelial dystrophy and corneal edema at normal levels of intraocular pressure (IOP). The endothelium is described as being "degenerate"; and with an abnormal Descemet's Membrane it extends down over the trabecular meshwork in the anterior chamber angle.[3,11]

Cogan-Reese or the Iris Nevus Syndrome, described in 1963[14] is characterized by the development of pigmented nodules or nevi on the iris which may be mistaken for malignant melanoma.[14,15] The surface of the iris becomes abnormal, described as matted or smudged, and the changes may include ectropion uveae and anterior chamber angle changes. Iris atrophy, however, is not seen, and high levels of IOP and severe glaucoma are rarely seen in this variant of the ICE Syndrome.[21]

Despite these differing characteristics the three conditions have many common features.[2,7] They are all more frequently described in women than in men, and in whites than in blacks, and they tend to be first diagnosed in young adulthood.[8] All three are associated with unilateral glaucoma, but the characteristic disk cupping and visual field losses may be found with either normal or high IOP.[2] No risk factors for the development of the conditions have been described, and there is no apparent genetic basis.[16]

Patients usually present to an ophthalmologist complaining of blurry vision or visual changes such as halos around lights. These symptoms usually indicate corneal edema and/or an attack of acute glaucoma. Patients themselves may notice the development of iris holes, sometimes described by the patient as a "second pupil" or the pupil going "lopsided". Pain in the eye is another, less common, reason for the patient to present to a doctor.[2] The decision about which category a patient is placed in depends on which of the major characteristics that patient displays. Iris holes, for example, suggest PIA, iris nodules suggest Cogan-Reese Syndrome and corneal edema with neither holes nor nodules is Chandler's Syndrome.

There is no definitive treatment for ICE Syndrome. Therapeutic efforts are, therefore, directed toward controlling the glaucomatous changes. Medical treat-

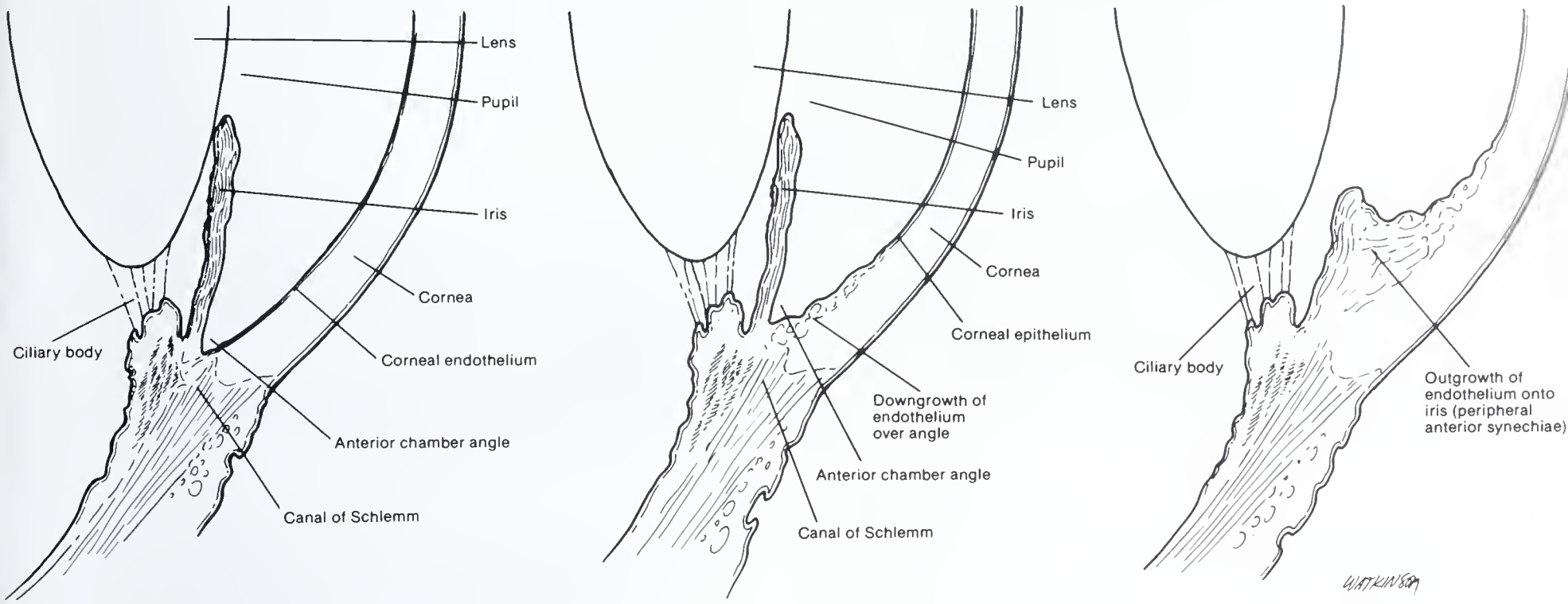


Figure 1: Cross section of eye showing anatomy and changes in the anterior chamber angle as peripheral anterior synechiae forms.

ment is the first line of therapy, as with chronic open angle glaucoma,[8] but surgery is resorted to when necessary, and has been reported to be relatively successful.[22]

The differential diagnosis of the changes seen in the ICE Syndrome is quite long.[2] The corneal endothelial changes must be differentiated from other dystrophies such as Fuchs' dystrophy (endothelial changes and atrophy associated with epithelial and stromal changes) or posterior polymorphous dystrophy (an autosomal dominant disease where the endothelium undergoes metaplasia to an epithelial form.)[17] The iris changes could be confused with aniridia (the congenital lack of an iris), with malignant melanoma (a primary cancer of the pigment cells of the iris), with neurofibromatosis (von Recklinghausen's disease - hyperplasia of nerve fibers), with posterior polymorphous dystrophy, or with inflammatory changes. It is beyond the scope of this paper to discuss the diagnostic features of each of these conditions, but it is important to make a correct diagnosis since treatment, where possible, differs for the various conditions.

There are many common features between the three variants and patients may progress from one subtype to another. A number of authors have, therefore, suggested that this indicates that the three variants are a spectrum of one disease with a common pathogenesis.[2,4,7]

Pathogenesis

The pathogenesis of the three components of the ICE Syndrome was for many years a mystery. A review by Shields in 1978[2] summarized the early attempts to explain the changes seen in this condition. One theory suggested that vascular insufficiency led to iris ischemia resulting in the stromal loss. Fluorescein angiography of the iris has shown vascular abnormalities, but these are now felt to be secondary changes rather than the primary defect. Two other early theories suggested that inflammatory changes, or a primary iris abnormality may be the cause of the syndrome, but these have not been supported and are no longer held.

The unifying feature between the subtypes, and thus the primary abnormality, is considered to be changes in the corneal endothelium, visible on light microscopic and electron microscopic examination. Recent studies have shown the endothelial cells to be larger than normal, and to show abnormal evidence of increased mitotic and migratory activity. There are also gaps in the endothelial membrane, the intercellular borders are abnormal, and many cells are nonfunctional and necrotic. The product of those cells which can synthesize also differs from that produced by cells in a normal membrane.[4,10,18] The endothelium shows a loss of contact inhibition as it migrates from the posterior cornea across the angle of the anterior chamber and the trabecular meshwork and onto the iris, forming peripheral anterior synechiae (PAS). This migration and PAS formation is considered to be the primary cause of the abnormalities seen clinically.[2,3,4,11]

A synechia is a pathological fusion of two elements in the eye that are normally separate, in this case between the back of the cornea and the iris. In progressive iris atrophy, the formation of the synechiae tethers the iris down. When two or more PAS form at different locations around the iris, contraction of the iris puts tension onto it which eventually leads to rupture of the delicate iris stroma and hole formation.[1,4] Between the areas of endothelialization, however, the iris is normal, indicating that iris changes are not the primary problem.[4]

The development of unilateral glaucoma is also explained by this theory. The growth of the endothe-

lium across the angle and the trabecular meshwork blocks the Canal of Schlemm and prevents the outflow of aqueous. This results in buildup of aqueous and increased IOP, thus leading to the characteristic optic disk changes and visual field losses seen in glaucoma. The failure of surgery to control the IOP can also be ascribed to the growth of membrane across the channels made to drain the aqueous.

According to this theory, the signs and symptoms of Chandler's Syndrome are considered to result when the endothelium is even more abnormal. It is, therefore, unable to sustain the growth and movement necessary to grow out over the iris and form synechiae, although endothelialization of the trabecular meshwork and the anterior chamber angle does occur. The endothelium is also, therefore, unable to maintain the corneal stroma in the manner to which it has been accustomed, thus resulting in corneal decompensation and edema even at low values of IOP.[11]

The third variant of the ICE Syndrome - the Cogan-Reese or Iris Nevus Syndrome - results when the downgrowth of the endothelial membrane onto the iris "pinches off" small islands of iris, thus forming the characteristic nevi. The nevi are a marker of endothelialization, and occur where the stroma is flat and/or effaced near synechiae or ectropion uveae.[1]

Although the overgrowth of the endothelium results in the signs and symptoms of the different subtypes of the ICE Syndrome, a recent study used a specular microscope to examine the endothelium of the posterior cornea.[10] As the result of their observations, they suggested the subdivision of ICE into two groups with two subgroups each, rather than into the three standard variants which are based on gross appearance. The authors suggested that the subdivision of patients by microscopic categorization is more useful than that provided by the classical definitions of PIA, Chandler's Syndrome, and Cogan-Reese Syndrome for two reasons. Firstly, the classical variants may be difficult to differentiate, and patients may progress from one form to another. Secondly, their grouping gave them the ability to predict which patients were likely to develop increased IOP, which is important from both a management and a prognostic point of view.[7] It is important to remember, however, that many authors have reported the presence of severe corneal edema in patients with normal IOP.

Now that it is realized that the overgrowth of an abnormal corneal endothelium is responsible for the signs and symptoms of the ICE Syndrome, the logical question becomes: Why should the endothelium so misbehave? Endothelialization can occur secondary to trauma such as contact between different elements in the eye, or secondary to inflammation. In the ICE Syndrome, however, the cause of the changes is yet to be determined. The fact that the condition is almost always unilateral suggests that the condition is acquired, and electron microscopic studies support this. The portion of Descemet's Membrane which is laid down by the endothelium in prenatal life (the Anterior Banded Zone) differs noticeably from that produced after birth (the Posterion Non-banded Zone). Examination of surgical specimens of the cornea by EM show a normal prenatal membrane with the abnormalities beginning some years after birth, but also some years before the onset of clinical symptoms.[19,20] These facts have recently led some authors to consider a viral infection as a possible cause for the endothelial transformation but no support for this theory has yet been reported.[19]

The ICE Syndrome is a fascinating clinical phenomenon with an equally interesting pathological basis. Much more is known about it now than even a few years ago, but much more remains to be elucidated.

Acknowledgements

I would like to thank Dr. Cain Kirkness and Dr. Linda Ficker for their help in the research for and writing of this article.

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Answers to Quick Dx-1 - Intestinal Obstruction

1. Case 1. Duodenal atresia - double bubble sign!
   Case 2. Hirschprung's disease
   Case 3. Malrotation

2. Green coloured emesis, no bowel movement within 24 hours of birth and abdominal distension
3. Case 1. duodenojejunostomy (side-side anastomosis)
   Case 2. temporary colostomy - definitive operation at one year.
   Case 3. if viable untwist and divide Ladd's bands; if not, resection.

Answers to Quick Dx-2 - CXR Quiz

1 - B: 40-50% left pneumothorax
2 - D: left mastectomy
3 - A: left lower lobe pneumonia and free air under right hemidiaphragm (iatrogenic)
4 - C: normal CXR

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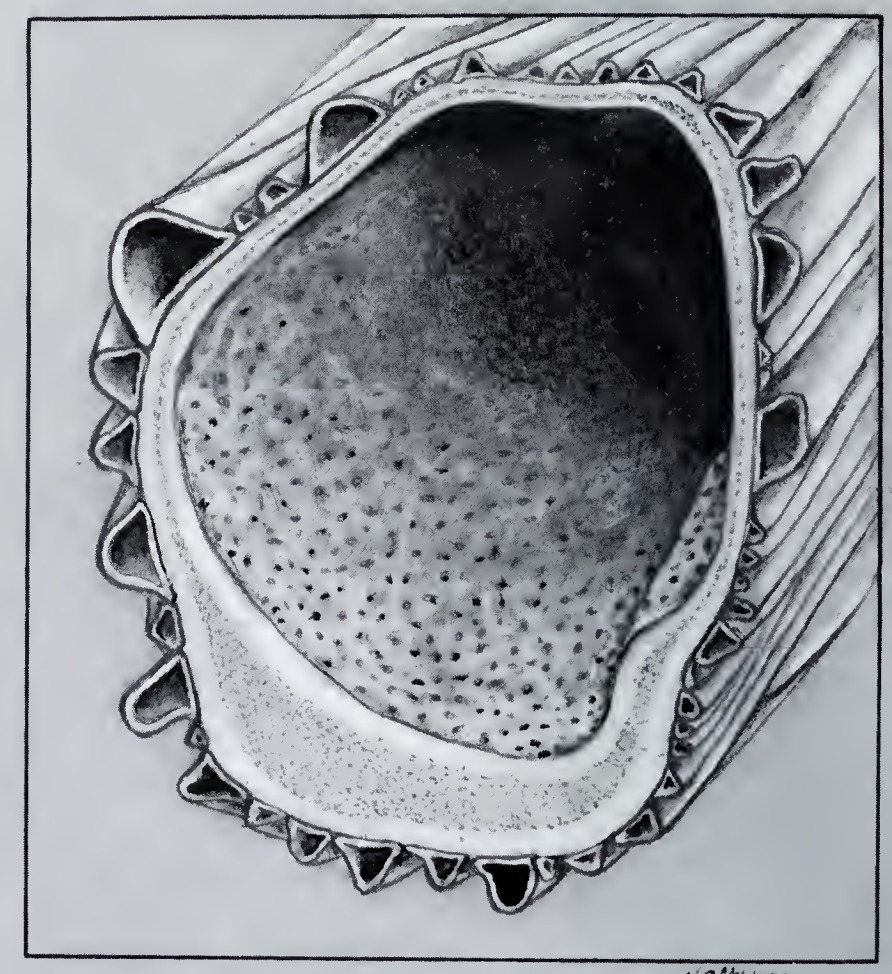


V.A. Gibbons

Hereditary Nephritis in Man and an Animal Model in Samoyed Dogs

*Filiberto Altomare, B.Sc. 9T1 and R. Baumal, M.D., Ph.D.**

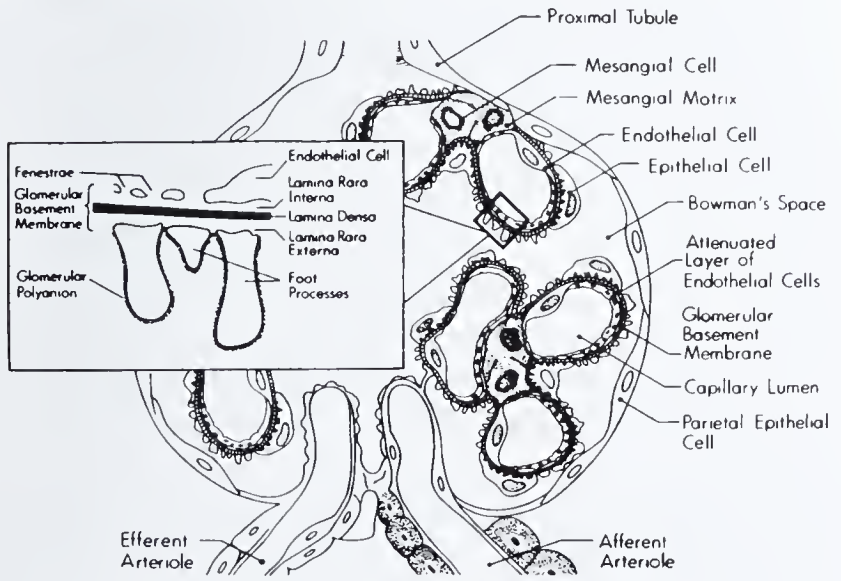
**Department of Pathology, Hospital For Sick Children*

Abstract

Hereditary nephritis (HN) in man resembles Samoyed hereditary glomerulopathy (SHG) in dogs. The initial clinical symptom of HN is hematuria, which may be associated with nerve deafness and various eye disorders. Males are affected more severely than females and are likely to progress to renal failure. Similarly in SHG, affected male and carrier female Samoyed dogs develop proteinuria, but only males deteriorate to end-stage renal disease. The pathologic lesion is located in the glomerulus and can be seen by electron microscopy. Immunofluorescence studies have identified a biochemical defect in HN and SHG. These and future studies suggest the animal model will be an essential investigative probe into HN in man.

Introduction

Kidney diseases may be classified according to the structure which is the primary site of injury. On this basis, there are numerous diseases which affect glomeruli, tubules, interstitium, and blood vesssels in the kidney. The glomerulus is the pivot of renal anatomy and physiology, and, consequently,diseases of glomeruli usually progress to involve the other three major components of the kidney. The glomerulus consists of four distinct cell types (endothelial, mesangial, and epithelial cells, and resident macrophages), a capillary wall (the glomerular basement membrane (GBM), and a supporting matrix and cells (mesangium) (Fig. 1). Injury to the glomerulus is characterized by one or more of four histologic abnormalities of these constituents: leukocytic infiltration, GBM thickening, hyalinization and sclerosis, and cellular proliferation. Cellular proliferation and extent of glomerular involvement in glomerular diseases may be used to classify glomerulonephritides (Table 1). Glomerular diseases can be either hereditary or acquired and may involve immunologic or nonimmunologic mechanisms of tissue injury. One glomerular disease, hereditary nephritis (HN), was first described by Alport in the early twentieth century in a family whose members developed renal failure and nerve deafness. Many descriptions have appeared since that time.[1,2] Hematuria is the cardinal feature, with males afflicted more severely than females, progressing to renal failure. Other associated symptoms include nerve deafness and ocular defects. Incidence of HN is very low, three percent of renal failure in children resulting from HN. There is a spontaneously occurring experimental model for human HN in Samoyed dogs that has proven invaluable in helping to elucidate the pathogenesis of HN. This paper will explore various aspects of the disease in man and the experimental model in dogs.



Modified from the New England Journal of Medicine

Fig. 1: Diagram of glomerulus illustrating the various cell types of the glomerulus except for resident macrophages. The inset gives a detailed depiction of the capillary wall.

Table 1: Classification of Glomerulonephritides

1. Non-Proliferative Glomerulonephritides: a. Minimal change glomerulonephritis (GN) b. Focal segmental glomerulosclerosis c. Membranous GN
2. Proliferative Glomerulonephritides: a. Focal proliferative GN b. Diffuse proliferative GN c. Membranoproliferative GN

A. Human Hereditary Nephritis

Clinical and Genetic Features

There appear to be various forms of HN, based on the age of development of renal failure and the presence of extra-renal dysfunction (Table 2).[3] Patients with HN classically demonstrate renal dysfunction in association with various extra-renal lesions such as sensorineural deafness (referred to as Alport's syndrome) and lens abnormalities, particularly anterior lenticonus (i.e., thinning of the lens capsule and protrusion of the lens substance into the anterior chamber of the eye). In childhood, the most frequent renal symptom is hematuria (microscopic or gross, persistent or recurrent). Proteinuria and/or nephrotic syndrome may develop after a number of years. Affected males often progress to end-stage renal disease (ESRD) by the third decade of

life but renal failure may even occur in childhood. Affected females with HN often develop a milder form of the disease. At present, no specific treatment is available to ameliorate the course of HN and dialysis and/or renal transplantation are the only means for managing the ESRD which eventually develops.

Table 2:
Characteristics of five phenotypes of Hereditary Nephritis

Type	Age at ESRD	Deafness	Other Defects	Inheritance
I	Juvenile	Yes	Ocular	X-linked
II	Adult	Yes	None	X-linked
III	Adult	No	None	X-linked
IV	Unknown	Yes	Thrombocytopathia	Autosomal
V	Juvenile	Yes	Ocular	Autosomal

Pathology of the Kidney

Early in its course, examination of the kidney by light microscopy (LM) shows normal glomeruli. However, at a later stage in the disease, glomerular abnormalities are seen, including increased mesangial matrix, mesangial hypercellularity, thickening and splitting of glomerular basement membranes (GBM) and glomerulosclerosis. Examination of glomeruli by immunofluorescence (IF) shows no deposition of immunoglobins or complement. Electron microscopy (EM) of glomeruli reveals multilaminar splitting of the lamina densa portion of GBM with electron-dense particles between the split layers (Fig. 2). If splitting is widespread, the changes seen by EM are pathognomonic of HN. However, some patients with HN may show a normal or thin GBM rather than one which is split.

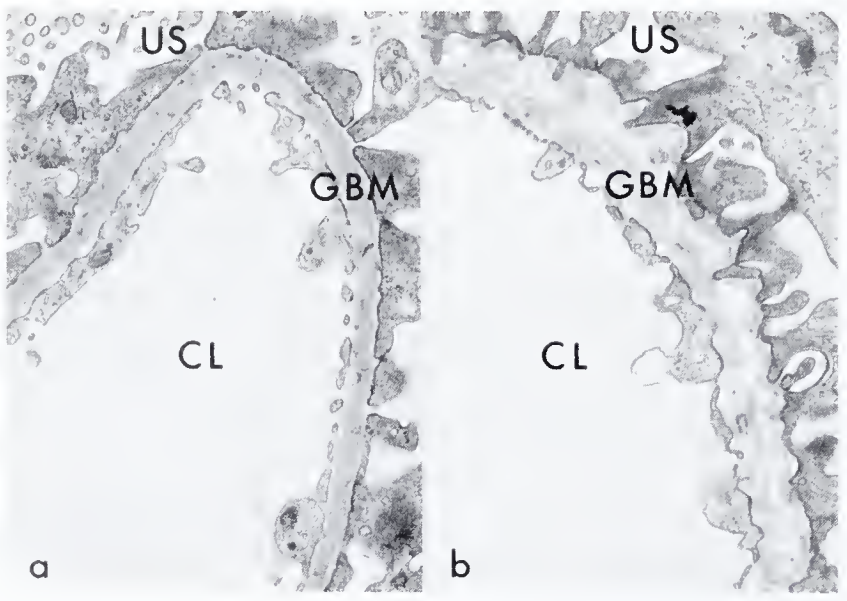


Fig. 2: EM of glomerular capillaries of a) a normal person and b) a patient with HN. In a) the GBM is not split, in contrast to b), where there is multilaminar splitting with scattered electron-dense particles. (US = urinary space, CL = capillary lumen, GBM = glomerular basement membrane).

Proposed Pathogenesis

Insight into the pathogenesis of HN has been gained from IF studies using serum obtained from individuals who suffer from Goodpasture's syndrome.[4] These sera contain antibody which is reactive with what has been referred to as the Goodpasture antigen (GPA) component of GBM. Recent biochemical studies have shown that GPA is contained in the NC1 domain at the carboxy-terminal end of the collagen type IV molecule.[5] When glomeruli from renal biopsies obtained from non-HN patients were examined by IF using Goodpasture's serum, staining of GBM was seen (Fig. 3a). However, the GBM of most males and some females with HN failed to stain, suggesting that there was a defect in the production or processing of GPA in HN (Fig. 3b). This finding has been supported by the observation that, when the NC1 domain of collagen type IV was extracted from glomeruli of kidneys of a few patients with HN, it was abnormal, as detected by gel electrophoresis. Further studies on pathogenesis have not previously been carried out because no suitable experimental model for HN had been described.

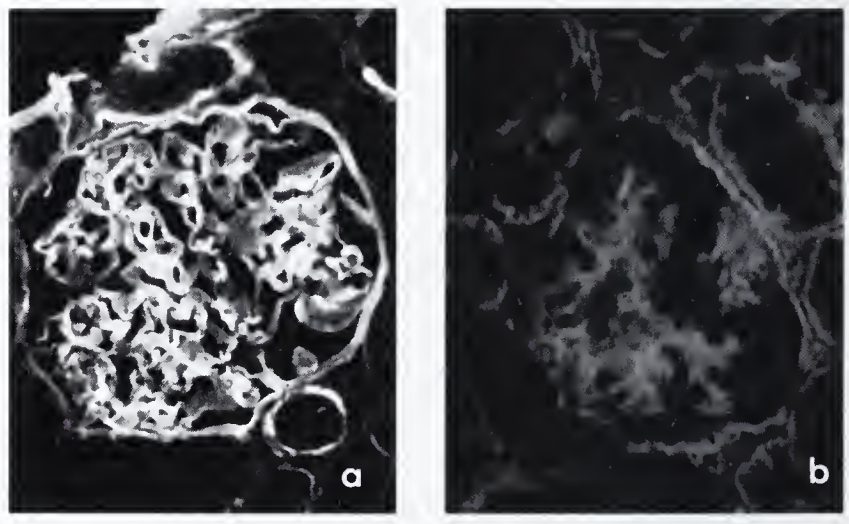


Fig. 3: IF microscopy using Goodpasture's serum of glomerulus of a) a normal person and b) a patient with HN. There is positive staining of GBM in a but not in b.

B. Samoyed Hereditary Glomerulopathy (SHG) in Dogs As a Model For Human HN

Clinical, Genetic, and Pathologic Features of SHG

A family of Samoyed dogs was accidentally found in which 50% of the males died of renal failure between 8-10 months of age, while the remaining 50% lived a normal life span. In addition, 50% of the females were carriers of SHG, while the remainder were normal.[6] This pattern is consistent with an X-linked mode of inheritance, similar to X-linked forms of human HN. SHG resembled X-linked HN in two additional aspects. First, examination of GBM of affected male dogs with SHG by EM showed extensive multilaminar splitting.[7] In contrast, carrier females showed only focal areas of splitting. Second, the NC1 domain of the collagen type IV molecule could not be demonstrated by IF in GBM of affected males with SHG using human serum containing anti-GBM antibody. In contrast, the NC1 domain was detected in GBM of unaffected dogs.[8] A defect in NC1 domain could also be demonstrated by IF in a number of extra-renal basement membranes (i.e., lens capsule, retina, basilar membrane of inner ear, lung, choroid plexus, and skin), even though no abnormalities could be detected by EM in these various basement membranes.

What We Have Learned About Human HN From the Animal Model

In both human HN and SHG, glomeruli fail to stain by IF when treated with Goodpasture's serum, pointing to an abnormality of the NC1 domain of collagen type

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IV in both conditions. NC1 was isolated from GBM of unaffected and affected male Samoyed dogs and examined by gel electrophoresis. NC1 in GBM of unaffected dogs showed 3 monomer bands (24 kD, 26kD, and 28kD) and dimer bands at approximately 46kD (Fig. 4, gel A). In contrast, NC1 in GBM of affected male dogs showed an absence of the 24kD monomer band (Fig. 4, gel B). Next, NC1 in GBM of the dogs was examined by Western blotting, a technique in which the bands which were resolved by gel electrophoresis were blotted onto nitrocellulose paper, which was then treated with anti-GBM antibody. Western blotting of NC1 in GBM of unaffected dogs showed binding of the antibody to the dimers and the 24kD and 26kD monomers (Fig. 4, gel C). However, in the case of NC1 in GBM of affected male dogs, binding was seen only to dimers (Fig. 4, gel D), indicating that there was a defect in the 26kD component, as well as in the 24kD portion demonstrated above. In further studies, it has been determined that the defect in NC1 in GBM of affected male dogs was present at birth, suggesting that SHG, and presumably human HN, were congenital diseases. Further studies are aimed at attempting to define more precisely the defect in NC1 in GBM and other basement membranes of affected male dogs and to relate this information to human HN.

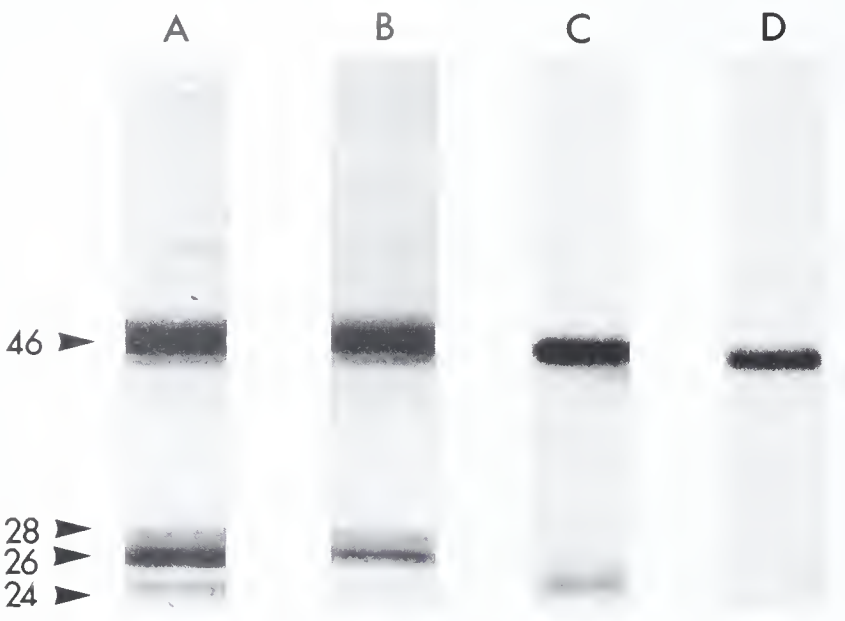

Fig. 4: Gel electrophoresis of NC1 in GBM of a) unaffected and b) affected male dogs. Western blotting using anti-GBM antibody of NC1 in GBM of c) unaffected and d) affected male dogs are also shown. Note that the 24kD and 26kD monomer bands seen in c) are not present in d).

Summary and Perspectives

Hereditary nephritis is a glomerular disease that progresses to ESRD primarily in affected males. Carrier females tend to express less severe symptoms. Assessment by immunofluorescence and electron microscopy has identified an animal model for HN. This has proven vital in determining the pathologic defect: the 24kD portion of NC1 domain of collagen type IV of GBM is absent and the 26kD component has altered antigenic determinants. The altered NC1 domain is present at birth. Work is being directed at elucidating the genetic basis of the disease and at determining the biochemical integrity of NC1 domain in extraglomerular basement membranes in unaffected and affected Samoyed dogs. Of interest is the comparison of any differences with the NC1 defects detected in GBM. Finally, future work is looking into treatment strategies such as dietary restriction for HN-affected individuals.

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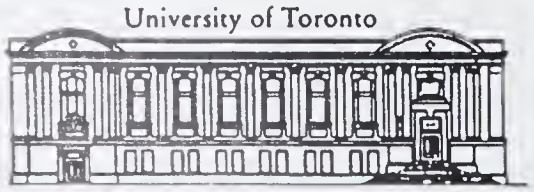

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A Comparative Review of the Magnuson-Stack and Putti-Platt Operations for Recurrent Anterior Dislocation of the Shoulder

Ato Sekyi-Otu, 9T0, Michael West, M.D., F.R.C.S.(C)

Abstract

A comparative functional analysis of 18 operative cases for repair of recurrent anterior shoulder dislocations, consisting of 9 patients who underwent the Magnuson-Stack procedure and 9 patients who underwent the Putti-Platt procedure at a Toronto community hospital, was performed. Range of motion, strength, stability, pain, relative function and complications were assessed in all patients with an average follow-up of 39 months. Pre- and post-operative x-rays were reviewed for Bankart and Hill-Sachs lesions, and other degenerative changes.

The data indicated that excellent results were obtained in 5 of the patients who underwent the Magnuson-Stack procedure, and in only 2 of the patients who underwent the Putti-Platt procedure. Range of motion and strength were most limited in the Putti-Platt group. While Bankart and Hill-Sachs lesions were not consistent pathological findings in either operative group, subscapularis laxity was a suggested common cause of recurrent shoulder instability in both groups. Overall analysis of results favours the Magnuson-Stack repair over the Putti-Platt in this series.

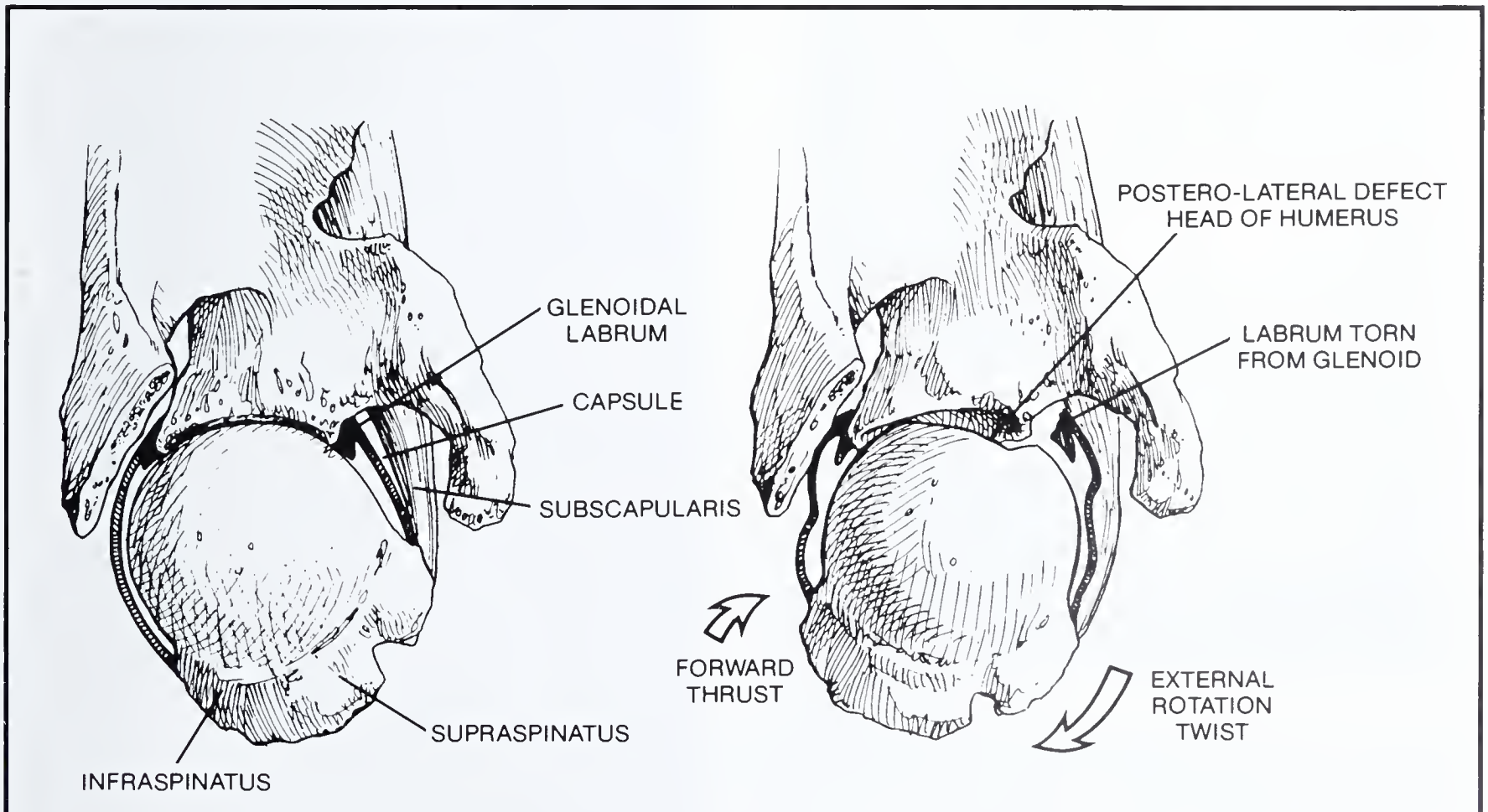

Figure 1a

Figure 1b

Figure 1a: The glenohumeral joint in a normal shoulder showing the fixed anterior stabilizers: the anterior capsule, glenoid rim and glenoid labrum. The subscapularis muscle is the main dynamic stabilizer.

Figure 1b: In recurrent anterior dislocations, the anterior capsule and labrum may be avulsed from the glenoid rim (Bankart lesion). The postero-lateral compression defect in the humeral head (Hill-Sachs lesion) results from repeated impaction against the glenoid rim (1).

The shoulder is more prone to acute and recurrent dislocations than any other joint in young adults. A number of anatomical and biomechanical considerations contribute to this predisposition. Anterior stability of the glenohumeral joint is determined by several structures. Fixed stabilizers of the shoulder anteriorly include the anterior capsule, the glenoid labrum and the glenoid rim (Figure 1a).[1] The glenoid cavity itself is relatively small compared to the humeral head thus providing little stability. The glenohumeral ligaments contribute little to joint stability. The subscapularis muscle is the chief dynamic anterior stabilizer. The shoulder is therefore a very mobile joint with minimal bony stability.[2]

Acute anterior dislocations of the shoulder are commonly seen in athletes as the shoulder is forced into external rotation and abduction. The anterior displacement of the humeral head often results in the avulsion of the capsule and fibrocartilaginous labrum from the anterior rim of the glenoid cavity (Bankart lesion) (Figure 1b). Once the integrity of the capsule and the anterior labrum is lost, the shoulder joint is prone to recurrent anterior dislocations at greater frequencies and with less stressful external forces, particularly in young active males.[3]

The diagnosis of recurrent anterior shoulder dislocation is made clinically. History usually reveals an initial acute trauma to the shoulder. Subsequent subluxations or frank dislocations occur when the arm is abducted and externally rotated, as in throwing; the patient recalls the shoulder "giving way" on each occasion. On examination, the patient demonstrates the 'apprehension sign' when the arm is placed in this position.[3]

Radiographic identification of bony lesions may be used to confirm the clinical diagnosis when necessary. An impression fracture of the postero-lateral humeral head (Hill-Sachs defect) is a result of repeated impaction against the glenoid rim during dislocations. The Bankart lesion may present as an osseus or cartilaginous defect of the anterior glenoid rim[4,5] (Figures 2a and 2b).

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Figure 2a: Radiographic appearance of the Hill-Sachs lesion (arrow): the postero-lateral compression defect of the humeral head is commonly seen in recurrent anterior shoulder dislocators.

Over 150 surgical procedures for the treatment of recurrent anterior dislocation of the shoulder have been documented. Each operation was devised to correct the lesion that was felt to be the underlying cause of the condition. Repairs for Bankart and Hill-Sachs lesions, and subscapularis laxity have all been described. Among the relatively few operations in common use today that increase the dynamic stability of the shoulder are the Magnuson-Stack operation for lateral advancement of the tendon of subscapularis from its insertion on the lesser tuberosity to a more lateral position on the greater tuberosity, and the Putti-Platt operation in which the subscapularis muscle and the anterior capsule are both overlapped and tightened.[2]

The purpose of this study is to provide a comparative analysis of the Magnuson-Stack and Putti-Platt procedures, based on the evaluation of 18 patients.

Patients and Methods

Between 1980 and 1988, 18 separate cases of recurrent anterior dislocation of the shoulder underwent surgical management at North York Branson Hospital (9 Putti-Platt repairs and 9 Magnuson-Stack repairs). Surgery was performed in the dominant arm in 5 out of 9 patients in both groups. The male to female ratios were 6:3 in the Magnuson-Stack group and 8:1 in the Putti-Platt group. A history of trauma, either due to sports or work-related injuries was documented in 7 Magnuson-Stack patients and 8 Putti-Platt patients. A congenital etiology was documented in 2 Magnuson-Stack patients and 1 Putti-Platt patient; the condition was bilateral in each of these patients. The average age at first dislocation was 19 and 20 years respectively, and at surgery was 22 and 27 years respectively.

Figure 2b: Radiographic appearance of the Blankart lesion (arrow): avulsion of the glenoid labrum may present as an osseus or cartilagenous defect of the anterior glenoid rim.

Post-operative follow-ups were conducted through a combination of personal and telephone interviews and chart reviews. The average follow-up period for the Magnuson-Stack group was 29 months; and for the Putti-Platt group was 43 months. Range of motion was assessed by means of physical examinations and questionnaires involving various activities (for example, ability to throw a ball or to place the hand behind one's back or head). Pre- and post-operative shoulder strengths were assessed objectively through physical exams and documentation in charts, and subjectively through each patient's personal testimony. Shoulder stability as judged by the number of post-operative dislocations, and relative function, as judged by return to work and pre-operative activity levels, were also documented through personal questionnaires. Pain relief was subjectively assessed in interviews; complications were documented from chart reviews. Available pre- and post-operative radiographs were reviewed for obvious Bankart and Hill-Sachs lesions.

Grading of these results was adapted from the classification of Karadimas et al.[6] Patients with painfree shoulders, full stability and power, and full range of motion including over 90 percent of external rotation were considered to have excellent results. Shoulders in which either the range of motion was less than 90 percent of normal in any direction or in which the patient complained of pain and/or a feeling of instability were classified as satisfactory. Patients who experienced any post-operative dislocations had unsatisfactory results.

Operative Techniques

In the Osmond-Clarke technique for the Putti-Platt operation, the tendon of subscapularis is divided medial to its insertion on the lesser tuberosity, and the underlying capsule is incised. Next, with the shoulder internally rotated, sutures are used to tighten the anterior capsule. Finally, the medial division of subscapularis is advanced laterally and sutured onto the lateral division of subscapularis, assuring a stronger capsulomuscular wall (Figure 3a).

In the Magnuson-Stack procedure the shoulder is placed in internal rotation while the bony insertion of the subscapularis tendon is freed from the lesser tuberosity and stapled to newly wedged site laterally at the greater tuberosity (Figure 3b).

After either procedure, the shoulder joint is immobilized in adduction and internal rotation for 3 or 4 weeks. The patient is then encouraged to exercise the arm; at 6 weeks intense physiotherapy begins.[2]

Results

Obvious Hills-Sachs lesions were found in 3 of 4 patients receiving Magnuson-Stack repairs and in 3 of 7 patients receiving Putti-Platt repairs; no Bankart lesions were observed radiographically.

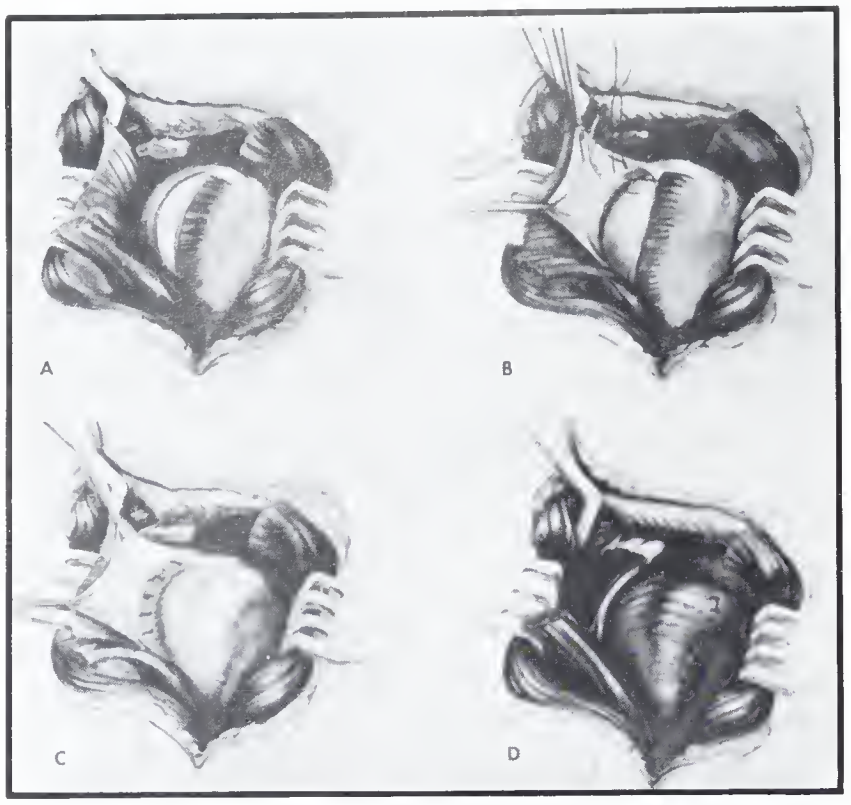

Figure 3a: Putti-Platt operation (technique of Osmond-Clarke). A. Subscapularis has been divided medial to its insertion on the lesser tuberosity, and the capsule has been incised. B and C. With the shoulder placed in internal rotation, sutures are used to tighten the anterior capsule. D. The medial division of subscapularis is advanced laterally and sutured to the lateral division of subscapularis (2).

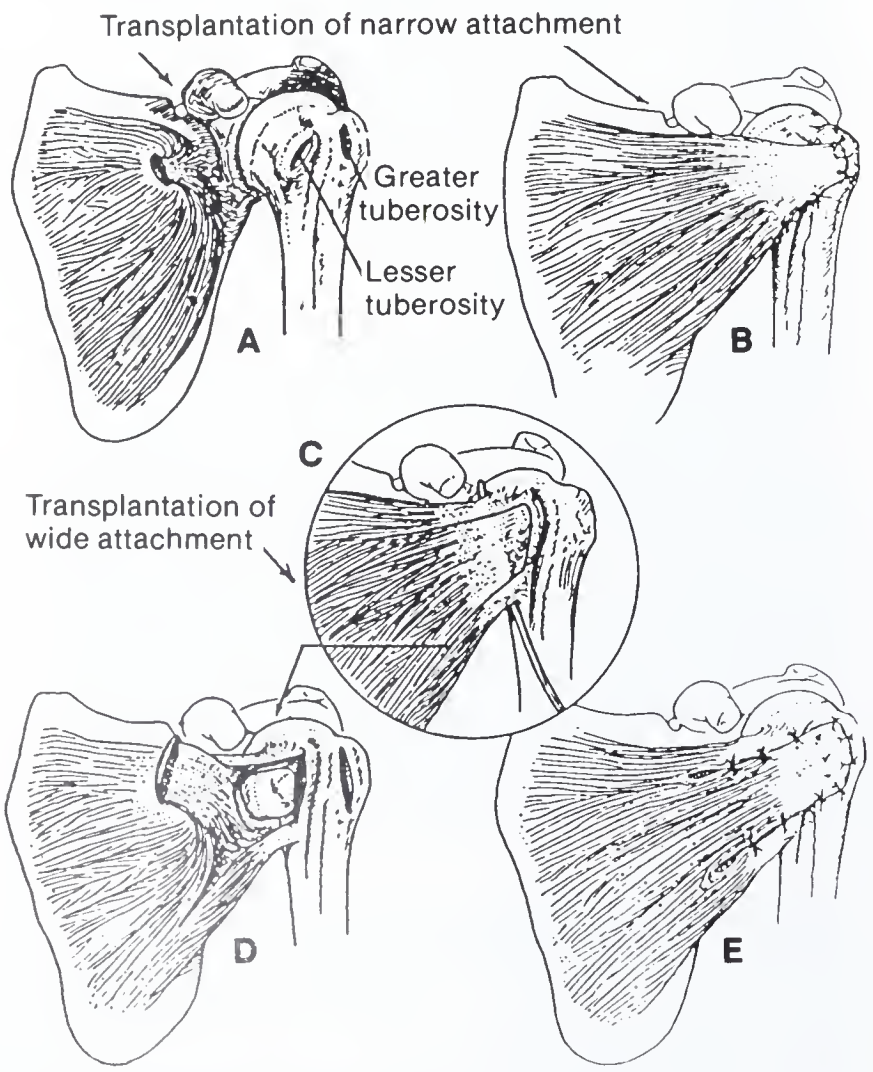

Figure 3b: Magnuson-Stack operation. The bony insertion of subscapularis is freed from the lesser tuberosity (A or D)and reinserted laterally in a wedge at the greater tuberosity (B or E), while the shoulder is placed in internal rotation (2).

Among the group of patients receiving the Magnuson-Stack repair, 8 patients reported complete return of range of motion in the shoulder, including full abduction and external rotation; one patient reported a limitation of external rotation greater than 10 percent. Among the Putti-Platt group, 4 patients reported complete return of range of motion while 5 patients reported limitation of external rotation of greater than 10 percent.

In both groups, 5 out of 9 patients were painfree at follow-up. One Magnuson-Sack patient experienced a dislocation after surgery; however, his shoulder is presently stable and has not required further surgery. One Putti-Platt patient has had three dislocations within three years following surgery, and is presently awaiting a second repair.

Six of the Magnuson-Stack patients demonstrated no loss of muscle strength; four of the Putti-Platt patients had no loss of muscle strength. One Putti-Platt patient demonstrated weakened biceps, diagnosed as a musculocutaneous nerve paresis. Another Putti-Platt patient experienced atrophy of the posterior third of his deltoid muscle, diagnosed as a circumflex nerve paresis; he went on to develop a reflex sympathetic dystrophy of his entire arm with the characteristic features of debilitating shoulder and hand pain, moist, hyperesthetic skin, edema, subcutaneous tissue atrophy, and disuse osteoporosis.

One subject (a Magnuson-Stack patient) developed a post-operative wound infection; this resolved with no sequelae. Post-operative results are summarized in Table 1.

According to the classification of Karadimas et al.,[5] Magnuson-Stack patients had excellent results, 3 had satisfactory results and 1 had an unsatisfactory result. Among the Putti-Platt Group there were 2 excellent results, 6 satisfactory results and 1 unsatisfactory result (Figure 4).

Table 1
Post-Operative Follow-up Results

	Magnuson-Stack (n = 9)	Putti-Platt (n = 9)
Complete Return of Range of Motion	8	4
External Rotation < 90% of Normal	1	4
Painfree Shoulders	5	5
Post-operative Dislocations	1	1
No Loss of Muscle Strength	6	4
	(n = 4)	(n = 7
Hill-Sachs Lesions	3	3
Bankart Lesions	0	0

Table 1: Table 1 lists the post-operative follow-up results of each operative group. Figures indicate the number of patients in each group that demonstrated a specific result.

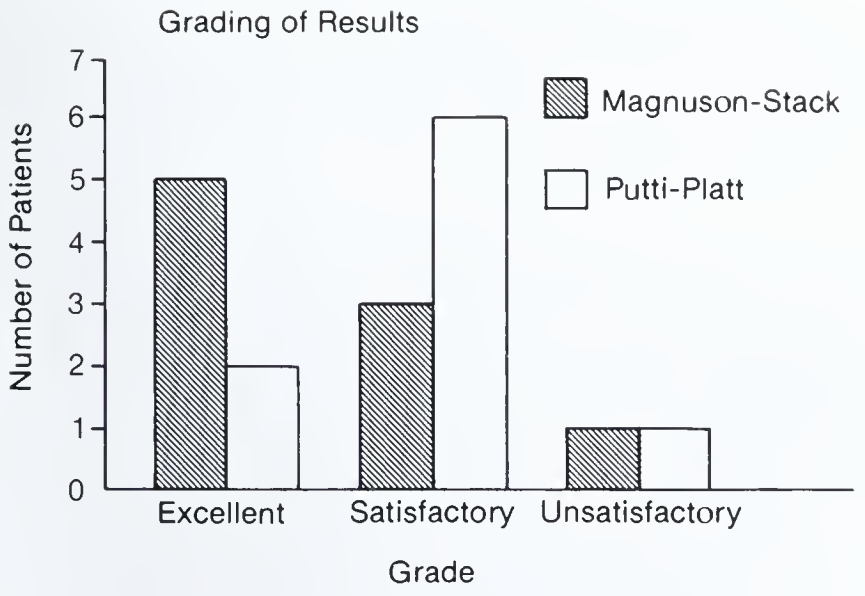

Figure 4: Grading of results was based on the classification of Karadimas et al. (6) (see text).

Discussion

The patients in this series were not unlike those of other studies with respect to age and sex distribution, and other etiological factors associated with recurrent anterior shoulder dislocations. Karadimas et al. reported an average age at surgery of 23 years in a study of 154 shoulders, although all patients in that study were male.[6] Caughey and Grey reviewed 50 shoulders in which the male to female ratio was 3:1.[7] In a study of 39 shoulders by Rowe et al., 75 percent had an antecedent history of trauma.[8] Atraumatic causes, suggesting congenital abnormalities such as glenoid dysplasia of ligamentous laxity have also been reported[9,10]; in these studies, instability was reported to be bilateral, as was observed in 3 patients in this study.

Seventy-five percent of the available x-rays of patients who underwent the Magnuson-Stack procedure showed Hill-Sachs lesions compared to 43 percent among the group of patients who received the Putti-Platt procedure and 55 percent for both groups combined. These percentages of Hill-Sachs defects in this series were similar to the 65 percent that was documented by Weber et al.[9] In Ahmadian's study of 38 shoulders, 50 percent had Hill-Sachs lesions while 78 percent had Bankart lesions.[11] Differences among various studies may be due to various diagnostic imaging views used to demonstrate such lesions.[4,5]

Neither of these defects has been shown to be the principle lesion accounting for the recurrence of dislocation. In particular, the original Bankart operation which corrects the Bankart lesion has been associated with a high incidence of post-operative dislocations.[12]

In a study by Symeonides, the role of the subscapularis muscle in maintaining anterior stability in the shoulder was investigated.[12]He found that with operations such as the Putti-Platt and Magnuson-Stack, in which the subscapularis muscle is tightened, there were much lower incidences of post-operative recurrences. Furthermore, Symeonides' observation of macroscopic and microscopic evidence of muscle laxity supported his own suggestion that a loose subscapularis muscle was a primary pathogenetic factor in recurrent anterior dislocations. The low incidence of recurrence after surgery in both the Magnuson-Stack and Putti-Platt groups in our series are consistent with his theory.

In this series, the Magnuson-Stack group appeared to regain greater mobility than the Putti-Platt group. Surprisingly however, there was no difference in the number of painfree patients in either group; apparently the introduction of a staple into the shoulder joint as used in the Magnuson-Stack repair made no comparable difference to pain development. In both groups, pain was produced by overuse of the joint and presented with classical arthritic symptoms, such as onset with 'damp weather'. Muscle strength was clearly more limited in Putti-Platt patients. The fact that the fibres of subscapularis are severed during this operation while they are kept intact in the Magnuson-Stack repair may account for this finding. Apparent intraoperative neurological damage leading to later complications in two Putti-Platt patients also contributed to this difference.

Conclusions

In this series, range of motion and power were most limited in the Putti-Platt patients. No comparable difference in chronic pain was found between the two groups. Relative function, as judged by the patient's ability to return to work and pre-operative activity levels, was most improved among the Magnuson-Stack group. Although Bankart and Hill-Sachs lesions were not consistent findings in either group, the low incidences of post-operative dislocations provided support to the theory proposing that the subscapularis plays a major role in the pathogenesis of recurrent anterior shoulder dislocations. Our results suggest that the Magnuson-Stack repair is superior to the Putti-Platt repair.

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Voice Change and Vocal Cord Paralysis

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History:

A 45 year old gentleman was seen by an otolaryngologist in September of 1983, for investigation of hoarseness and a "raspy voice". These symptoms had apparently followed an upper respiratory tract infection which had occurred nine months earlier. During this time, his voice had improved slightly for short periods but over all remained hoarse, raspy and tired easily.

On inquiry, the patient was not a diabetic. He admitted to smoking a package of cigarettes and to consuming half a litre of alcohol daily for the past thirty years. There was no difficulty speaking or breathing, and he denied any history of otalgia, dysphagia, odynophagia or weight loss. The remainder of the functional inquiry was negative.

Examination of the ears, nose and throat was normal except for indirect mirror laryngoscopy where a paralysis of his left vocal cord in the paramedian position was noted. There were no laryngeal mucosal lesions. There were no abnormal masses palpable in the neck, and the thyroid was not enlarged.

After a thorough work up, to exclude lung and thyroid malignancy, the left cord paralysis was attributed to an idiopathic neuropathy, likely viral, involving the left recurrent laryngeal nerve. It was decided to wait 3-6 months to see if any spontaneous recovery of cord movement occurred. When it did not, teflon vocal cord augmentation of the left (paralysed) side was performed with considerable improvement in the voice.

Four years later he presented to Sunnybrook Medical Centre complaining of left sided facial pain, dysphagia and sore throat of nine months duration. The facial pain was described as sharp in character and was located below the angle of the left jaw and up in front of the left ear. The dysphagia was worse with solids than liquids. Although there had not been a change in his appetite, he had lost 15-20 lbs. over the last year. he complained of fatigue and decreased energy. There was no difficulty with speaking or breathing, or any history of diasphoresis, flushing, diarrhea or hypertension. There was no family history of neck masses or of pheochromocytoma.

On examination, the patient was hoarse but in no apparent distress. Otic, nasal and nasopharyngeal examination was normal. The oral cavity showed no mucosal lesions. The tongue showed a left hemiatrophy and on protrusion deviated to the left (Fig. 1). The right side of the soft palate appeared to elevate better than the left side (Fig. 2). The left tonsil was more prominent than the right but there was no ulcerative lesion seen. Indirect mirror examination showed a left sided vocal cord paralysis in the paramedian position with good compensation from the right cord. Neck examination revealed a 2 x 2 cm mass near the left parotid tail. The mass was non-pulsatile and no bruit was heard over it. There was weakness of the left sternomastoid and trapezius muscles. He was therefore considered to have palsies of the last four left cranial nerves.

What is your diagnosis?

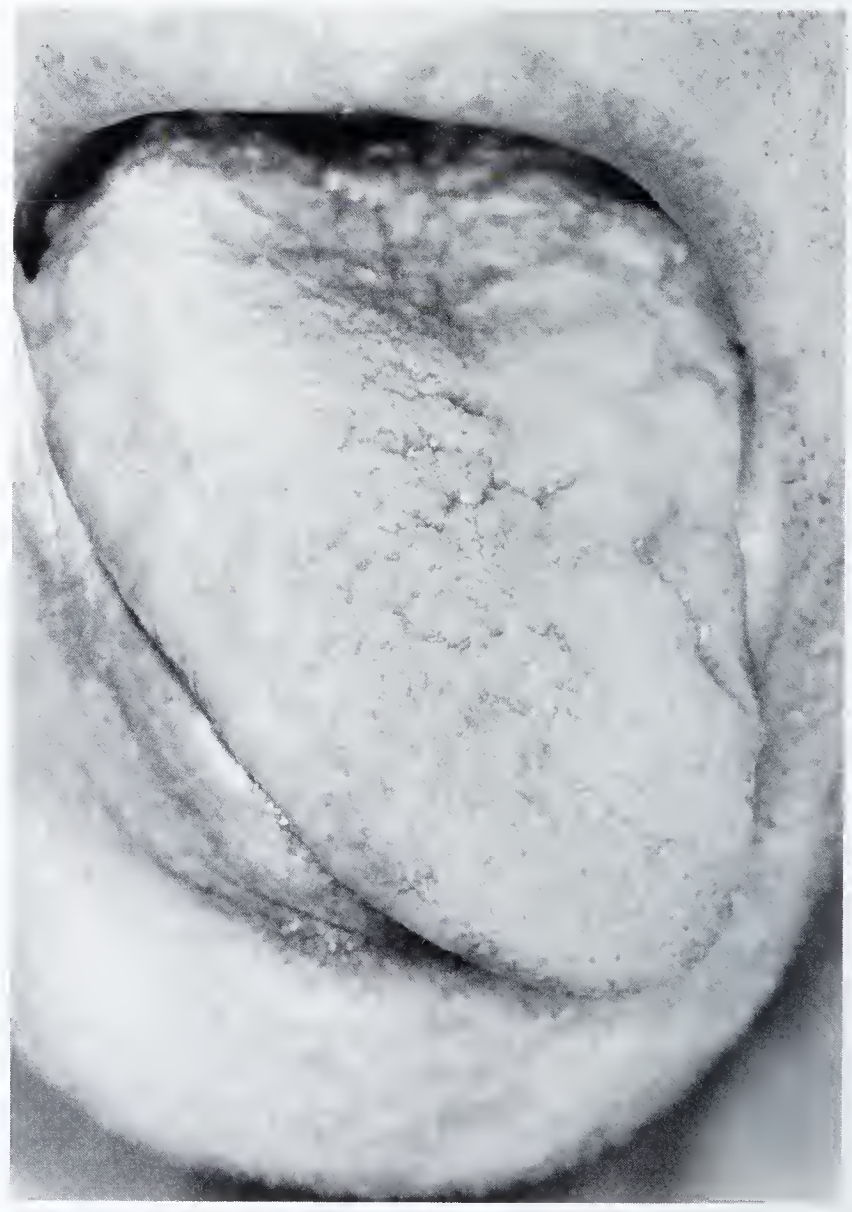

Figure 1: The tongue showed left hemiatrophy and deviated to the left on protrusion

TABLE 1

DIFFERENTIAL DIAGNOSIS OF VOICE CHANGE

VOICE CHANGE

CONGENITAL

1) Webs
2) Congenital paralysis
3) Cysts
4) Other

ACQUIRED

INTRALARYNGEAL DISEASE

1) Inflammatory
   - specific infections
   - allergic
   - irritants
2) Masses
   - benign
     vocal cord nodules
     vocal polyps
     contact ulcers
     reinke's edema
     neoplasms
   - malignant
3) Vocal chord paralysis
4) Vocal chord fixation
5) Trauma
6) Functional
7) Senile larynx

EXTRALARYNGEAL DISEASE

WITHIN THE NECK

Lesions produce voice change by:
1) direct pressure on the larynx
2) edema secondary to decrease venous or lymphatic drainage
   - trauma
   - peritonsilar abscess
   - thrombophlebitis of jugular bulb
   - tumors of the hypopharynx
   - other tumors of the neck

OUTSIDE THE NECK

These lesions affect the laryngeal nerves causing vocal cord paralysis

Figure 2: The right side of the soft palate elevated better than the left side

DISCUSSION: Voice Change

The differential diagnosis of hoarseness is listed in Table 1. Congenital and developmental abnormalities of the larynx are uncommon but do occur. The lesions usually present early in life with hoarseness or more seriously with airway obstruction. Among the more common abnormalities are congenital paralysis of the vocal cord, congenital cysts, laryngocele ventricularis, and congenital glottic webs. The latter are caused by the failure of normal separation of the true cord primordia. These webs are generally located in the anterior commissure. Other congenital lesions such as reduplication of vocal cords, and hypertrophy of various portions of the larynx have been reported but are very rare.

Inflammatory lesions of the larynx present with hoarsness in the setting of other symptom complexes, such as upper and lower respiratory tract infection, sore throat, or fever and chills. Inflammatory causes of hoarseness include a variety of specific infections of the larynx, allergic disorders and irritants. Viral upper respiratory tract infection may include the larynx and cause hoarseness, which might persist for weeks after the resolution of the infection. Bacterial infections of the larynx are not as common as viral infections. Beta-hemolytic streptococci or hemophilus influenzae are usually the offending organisms, and the patient is usually much more toxic and ill looking than with a viral infection. Bacterial epiglottitis may cause voice change early in the course of the disease, but then progresses to stridor and airway obstruction. Mycobacterial and fungal infections are rare and the hoarseness is preceded by

pulmonary symptoms which dominate the clinical picture. Irritants produce hoarseness by causing edema of the vocal cords and surrounding tissues. Irritants are usually inhaled or ingested, but may be autoirritants such as refluxed gastric acid or purulent secretions.

Vocal cord nodules, polyps and contact ulcers result from voice abuse. Vocal cord nodules are small masses of fibrous tissue that represent organized hematoma. These lesions characteristically develop at the junction of anterior and middle thirds of the free cords. Vocal cord polyps are a disorder of the middle aged and are more prevalent in females, particularly those that smoke and have jobs tht entail a lot of talking such as teaching or selling real estate. The polyps might involve the entire vocal cords, anterior 2/3, or may be more localized. The polyps are caused by edema in the subepidermal space. Contact ulcers are shallow and covered with a white or yellow membrane and are located at the tips of the vocal processes. Sometimes they are hypertrophic with granuloma formation.

Neoplasms may cause hoarseness by creating an irregular vocal cord border, or changing the mass or mobility of the cord. Benign neoplasms of the larynx are rare but have been reported. They include neuromas, chondromas, fibromas, and granular cell myoblastoma. Malignancy of the glottic region of the larynx, almost invariably squamous cell carcinoma in a smoker, usually presents with hoarseness, while superglottic carcinoma of the larynx is much more nonspecific presenting with odynophagia and referred otolgia. The rare subglottic carcinoma of the larynx presents with stridor and is sometimes misdiagnosed as asthma. A good rule of thumb is; hoarseness that is persistent for more than three weeks, particularly in a heavy smoker and drinker, requires visualisation of the larynx, and prompt referral to an otolaryngologist is recommended.

Vocal cord fixation is caused by disorders of the cricoarytenoid joints or the intrinsic muscles of the larynx. Cricoarytenoid joint fixation is usually due to rheumatoid arthritis or trauma during endotracheal intubation. The intrinsic muscles of the larynx become nonfunctional from invasion by neoplasms or an inflammatory process such as TB, sarcoid or syphillis. In rare cases laryngeal muscle disfunction is an expression of myasthenia gravis, in which case the voice gradually weakens through conversation.

Trauma produces hoarseness through edema, dislocation of arytenoids, or damage of the vocal cords with scarring.

Functional voice disorders are further classified into vocal fatigue, spastic dysphonia and dysphonia plica ventricularis. A speech pathologist will be able to help the physician by providing a more complete voice history, and aid in the diagnosis and management of these conditions.

Finally with old age the cords become bowed and produce a voice with a hoarse rough quality.

Vocal cord paralysis:

Vocal cord paralysis is a sign of disease, not a diagnosis, and requies a logical investigation along the vagus nerve and its branches. A review of the relevant anatomy of the vagal system will help in localizing the lesion (Fig. 3).

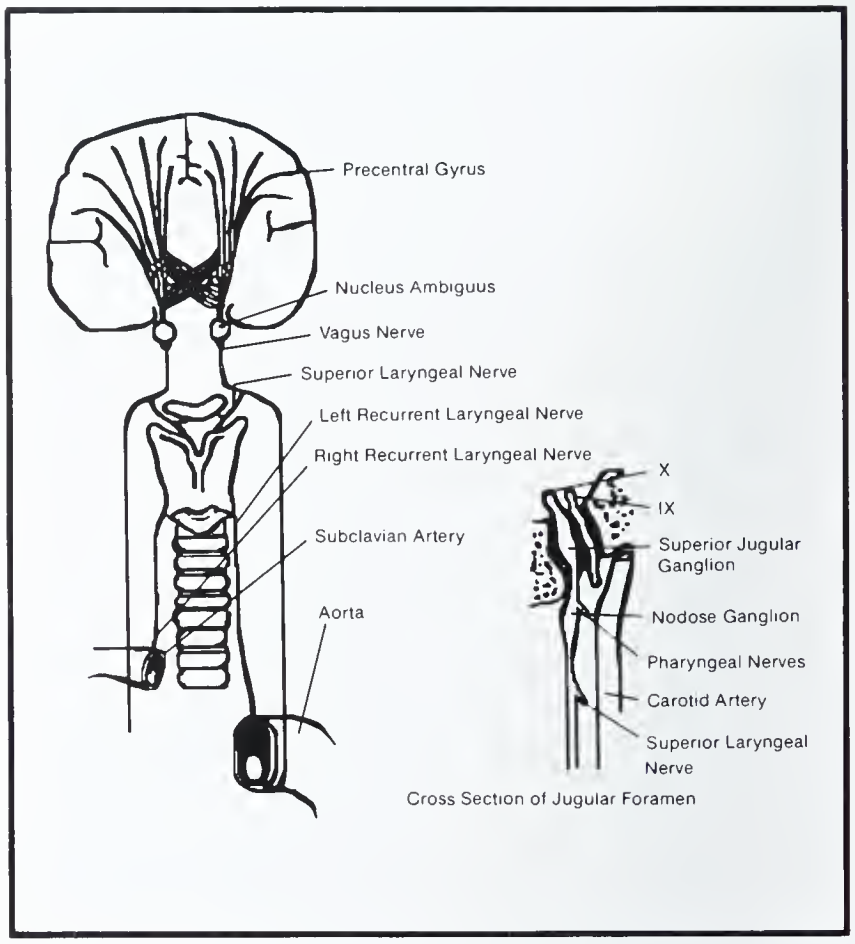

Figure 3: Anatomy of the vagal system

The supranuclear fibres originate in area 4 on the rostral side of the lateral fissure. These fibres travel through the internal capsule, cerebral peduncles and the pons to provide bilateral innervation to the vagal nuclei.

The medulla contains the nuclear centres of the vagus nerve. The nucleus ambiguus provides ipsilateral special visceral efferent fibres to muscles of the larynx, pharynx and upper esophagus. The nucleus solitarius receives special visceral afferents. The dorsal and inferior salivary nuclei supply and receive general visceral efferents and afferents. The respiratory and cough centres are found near the nucleus ambiguus.

The vagus nerve is formed by rootlets emerging from the brainstem. The nerve exits the skull through the jugular foramen and is associated with the jugular vein, CN IX, CN XI and CN XII. Above the nodose ganglion the vagus produces branches to the pharyngeal plexus. This pharyngeal plexus supplies the mucosa and muscles of the pharynx, and upper esophagous. Below the nodose ganglion the vagus produces the superior laryngeal nerve, which is motor to the cricothyroid muscle and sensory to the mucosa of the supraglottic larynx.

The vagus nerve continues into the chest. The left vagus nerve passes anterior to the aortic arch and gives off the left recurrent laryngeal nerve. This nerve lies lateral to the ligamentum arteriosum, loops around the aortic arch and ascends through the superior mediastinum to the larynx in the tracheoesophageal groove. After passing anterior to the right subclavian artery, the right vagus gives rise to the right recurrent laryngeal nerve, which loops around the artery and ascends to the larynx in the tracheoesophageal groove. The

recurrent laryngeal nerves are motor to the intrinsic muscles of the larynx (except cricothryoid muscle) and sensory to the subglottis of the larynx.

Lesions along the vagus nerve resulting in vocal cord paralysis can be divided into nuclear and peripheral.

Nuclear lesions of the vagus nerve produce a breathy voice and paralysis of the ipsilateral cord. The cough reflex is ineffective. Patients are also dysphagic and demonstrate achalasia of the cricopharyngeal muscle, because the lesion is above the pharyngeal nerves (Table 2).

TABLE 2

NUCLEAR LESIONS

Etiology

1. Congenital:	Arnold-Chiari, Klippel-Feil, Dandy Walker syndromes.
2. Vacular:	vascular thrombosis, A-V malformations, hemorrhage.
3. Inflammatory:	inferior bulbar poliomyelitis, Guillan-barre, diphtheria, rabies, tabes dorsalis, multiple sclerosis, tetanus, botulism.
4. Neoplasms:	primary and secondary.
5. Trauma:	accidental and iatrogenic.

Symptoms and Signs

1. Voice:	voice change
2. Cough:	decreased effectiveness with aspiration.
3. Swallowing:	dysphagia with cricopharyngeal achalasia.

TABLE 3

PERIPHERAL LESIONS

Etiology

1. Vascular:	basal hemorrhage, intracranial aneurysm, aortic aneurysm, left atrial hypertrophy.
2. Inflammatory:	meningitis, polyneuritis, diphtheria, inflammatory disease of the mediastinum, thyroiditis, neck abscesses, retroparotid syndromes.
3. Neoplasms:	neuroma, meningioma, glomus jugulare, chemodectoma, pulmonary neoplasms, esophageal neoplasms, laryngopharyngel lesions, lymphoma, thyroid carcinomas.
4. Trauma:	neck trauma, skull fracture.
5. Toxic causes	

Symptoms and Signs

1. Voice:	voice change
2. Cough:	ineffective with aspiration.
3. Swallowing:	usually not affected.

Peripheral lesions can be divided into high and low lesions relative to the nodose ganglion (Table 3). Both lesions cause a breathy voice with ipsilateral cord paralysis and an ineffective cough reflex with aspiration. In addition high lesions produce dysphagia because of the paralysis of the ipsilateral side of the pharynx. Physiologic compensation frequently occurs in low vagal lesions and the patient's voice and cough are not as badly disturbed. Concomitant lesions of CN X and CN XII are extremely suggestive of a lesion at the base of the skull.

90% of vagus nerve disorders are due to peripheral lesions. Approximately 60% of peripheral lesions are due to trauma and neoplasms. Trauma in the course of thyroid surgery remains the most common cause. The most common neoplastic cause is lung carcinoma constituting 50% of the cases of neoplasms. Despite careful evaluation paralysis of unknown etiology still constitutes a substantial group amounting to some 20% of vocal cord pasies. Viral neuritis is usually thought to be the cause and the incidence is considerably higher in males and involves the left cord more than the right. Fortunately, a large number of patients will have spontaneous spontaneous resolution of idiopathic paralysis within six months.

Diagnostic Assessment:

The workup of vocal cord paralysis determines the cause of neural loss and its position along the vagus nerve. This assessment includes a history, physical examination, laboratory tests, radiography and endoscopy (Table 4).

Patients with vocal cord paralysis should have a complete history with special emphasis on the time course of hoarseness, cough and dysphagia. Associated neurological symptoms such as hearing loss, vertigo, nystagmus, ataxia, and ipsilateral loss of facial sensation

TABLE 4

DIAGNOSTIC WORK UP OF VOCAL CORD PARALYSIS

History
↓
Physical Examination
↓
Blood count, VDRL, Blood sugar
↓
Chest X-ray
↓
Other Imaging Techniques including: Barium swallow, Thyroid scan, CT-scan
↓
Endoscopy
↓
Biopsy

with contralateral loss of pain and temperature are associated with nuclear lesions, while a history of neck trauma or surgery and symptoms of thyroid or lung neoplasms may be obtained in patients with peripheral lesions.

Although it is not usually possible to make a precise diagnosis simply by listening to the voice, it is usually possible to distinguish the rough quality of a lesion affecting the smooth border of the cord from the breathy air escape in the voice produced by an incompetent larynx such as in the case of vocal cord paralysis. A simple clinical test is to ask the patient to take a deep breath and count out loud. A normal person will count easily up to thirty or forty, while the patient with vocal cord paralysis will empty his chest and run out of voice after counting only up to ten or twelve.

The rest of the physical examination should include inspection of the larynx, examination of the neck for masses and neurological examination including special attention to the last four cranial nerves. Concomitant involvement of CN X and CN XI or CN XII is extremely suggestive of a high peripheral lesion because the course of CN XII and CN XI from the brainstem through the skull is entirely different from CN X while outside the skull they run in close proximity with CN X in the parapharyngeal space. Involvement of CN IX can occur in both nuclear and high peripheral lesions.

Laboratory studies should include blood count and VDRL; the blood sugar should be estimated because diabetics have higher incidence of idiopathic vocal cord paralysis.

The basic radiological workup for vocal cord paralysis is a chest X-ray, followed by a barium swallow and a thyroid scan. The chest X-ray rules out any lung, hilar or mediastinal disease, while the barium swallow may define the level of a vagal nerve defect, and also provides an assessment of the degree of aspiration. A CT scan is done instead of the barium swallow and the thyroid scan if the clinical presentation is suggestive or in the absence of clinical suggestion if the basic workup is all negative.

If any of the studies mentioned above shows any abnormality, further appropriate diagnostic tests are performed. Otherwise the patient is examined under anaesthesia; this includes quadroscopy and assessment of the mobility of the cricoarytenoid joints.

The patient presented here had hoarseness, dysphagia, palsy of the last 4 left cranial nerves and a mass near the tail of the left parotid gland. The patient was thought to have a high peripheral vagal lesion.

Radiological investigations in this patient included a chest X-ray, CT-scan of the head and neck and angiogram. The chest X-ray was normal. A non-homogenously enhancing parapharyngeal mass (5x2 cm.) with extension into the retrostyloid compartment was seen on CT-scan. An angiogram of the great vessels of the neck revealed a vascular mass high in left parapharyngeal space suppled by the external carotid especially the ascending parapharyngeal branch. The left internal carotid artery was found to be stenotic (Table 5).

An incisional biopsy produced tissue compatible with "chemodectoma".

TABLE 5

DIFFERENTIAL DIAGNOSIS OF A PARAPHARYNGEAL MASS

1. Salivary gland tumors
   - Benign mixed
   - Adenoid cystic carcinoma
   - Adenocarcinoma
   - Mucoepidermoid carcinoma
2. Neurogenic
   - Schwannoma
   - Neurofibroma
   - Chemodectoma
3. Miscellaneous
   - Branchial cleft cyst
   - Cystic hygroma
   - Hemangioma
   - Teratoma
   - Lipoma
   - Fibroma
   - Lymphoma
   - Parapharyngeal space abscess
   - Parapharyngeal adenitis
   - Metastatic squamous cell carcinoma

Chemodectomas are neoplasms that arise from paraganglion cells of the autonomic nervous system, and are neuroectodermal in origin. The most common chemodectomas of the head and neck develop in the jugular fossa and in the middle ear, however they may appear in the neck and the parapharyngeal space. Histologic staining techniques demonstrate catacholamines in low quantities in the paraganglia cells and vasoactive chemodectomas are at a higher risk of having a pheochromocytoma. Chemodectomas are usually benigh but malignant lesions have been reported. Vagal chemodectomas constitute 3% of all chemodectomas and commonly arise from one of the vagal ganglia (superior or nodose).

Chemodectomas may be removed surgically and this is the treatment of choice where it is pratical. The tumors are also responsive to radiotherapy with the usual behaviour after irradiation being that the lesion regresses partially but then remains stationary without further growth for lengthy periods up to 20 years or more. Radiotherapy is therefore an excellent alternative form of treatment where surgical excision is contraindicated by the size or location of the tumor or the risk of causing severe morbidity. Intraarterial embolization of the tumor is a useful preoperative adjunct and may reduce the hemorrhage from these very vascular tumors. The definitive treatment of tumors by embolization is still experimental. The patient reported here was treated with irradiation since it was felt that a surgical removal might have made his modest dysphagia much worse.

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The Medicine Chest: Health Care on Indian Reservations in Northwestern Ontario

Ian Park, 9T0

Amid the distressing poverty of Canada's poorest people stands a symbol of hope. Against disease and despair there are those who struggle.

Partly because they are the poorest people in our country, Native people also have the lowest standard of health. The life span of Native people is ten years less than the rest of Canadians. Morbidity is significantly greater in almost every category. Yet the situation would be much worse without the Nursing Stations and Native Hospitals, run by the Medical Services Branch of the federal Department of Indian Affairs. These facilities are part of the modern interpretation of the promise of the Medicine Chest.

Part of most of the treaties signed between the Canadian government and various groups of Native people is a commitment to provide a "Medicine Chest" to the Native people. Originally, it was, literally, a chest filled with medicines. But as medicine advanced, so too did the services provided to Native people of the reserves. Unfortunately, despite heroic efforts by the nurses and doctors providing the health care, the standards do not reach those enjoyed by the rest of Canadians. A brief exploration of the situation in the Sioux Lookout Zone will illustrate some of the reasons for this.

The Sioux Lookout Zone (SLZ) is a vast area of Northwestern Ontario stretching from Hudson's Bay to the Manitoba border, and lying north of the Canadian National Railway tracks. About 12,000 native people on 23 Reserves are served by the Zone Hospital, located in Sioux Lookout (about 250 km northwest of Thunder Bay), and by several Nursing Stations and their satellites, which are located on the Reserves themselves.

Two Native peple working at the Sandy Lake Nursing Station.

The University of Toronto has a special relationship with the SLZ, since it is partially responsible for the administration of the Zone Hospital and the provision of health care in the SLZ.

The Deer Lake Nursing Station.

There are four "levels" of care provided for the people on the Reservations. The most basic level, available to people on all Reservations (and the only primary care available to those living on the smaller Reservations), is provided by the CHR, or Community Health Representative. This is a Native person who has been trained for a variety of skills, including community health (audiology, immunization, etc.) and simple medical procedures (diagnosis and treatment or referral). They work out of the Satellite Clinics, which are usually small, one-room buildings with a single bed. Several Satellites are served from a more central Nursing Station, which is located in a larger nearby Reservation. The nurses who work at that central Nursing Station make periodic visits to the Satellites, to help the CHRs with more difficult diagnoses and treatments, including care of patients with more chronic diseases. Any patients too difficult for the CHR to handle are transported by air ("Medevac'ed") to the central Nursing Station.

The Nursing Station is the second level of care available. They serve the larger Reservations and the Satel-

lite Clinics. Two to six nurses live in residence at the Nursing Station (the size of the station, and the number of nurses stationed there, depends on the size of the community) and they perform most of the functions that one would normally find done by a physician in a more built-up area. These duties include prescribing (and dispensing) drugs. Doctors on staff at the Zone Hospital make periodic visits to the Nursing Stations to assist with the more difficult patients. As with the CHRs referring the most difficult cases, so the nurses can Medevac patients to the Zone Hospital if need be.

The Zone Hospital in Sioux Lookout is more than a hospital. It is the administrative center for all aspects of health care in the SLZ. A very important feature of this is the co-ordination of the transport of medical supplies to the Nursing Stations and Satellites, and administration of the Medevacs. Without the most modern (and expensive) communication and transportation facilities, it would be almost impossible to deliver health care to the Native people in the SLZ.

There are six to ten family physicians on staff at the Zone Hospital. As with the nurses at the Nursing Sta-

A treatment room at Deer Lake.

tions, physicians are expected to do much more than would normally be expected. Essentially, the "docs" have to perform as specialists in all areas. While there are occasional visiting residents in Pediatrics, Radiology, and so on, there is a chronic lack of specialist expertise. Again, the importance of communications become clear: physicians make their consultations mostly by telephone. When a patient's problem is too difficult, they are referred to the fourth level of the health-care system: they are flown to Thunder Bay, Winnipeg, or Toronto for definitive diagnosis and treatment.

From the above, it should be clear why the health care available to Native people is less than the national level. Other problems include the great cost, differences in culture and language, and "burnout" of nurses and physicians under the overwork, stress, and isolation.

The Native people of the SLZ also realize that the health-care system is less than it should be. In response to this, an inquiry under the Reverend Ted Scott is now studying the whole health-care system in the SLZ.

Perhaps the most important thing one can learn

from visiting the SLZ is that health depends on a great deal more than medical services. Poor hygiene, poverty, unemployment, and despair all contribute strongly to the poor health of Native people.

Yet there is reason for great hope. Native people are becoming better educated and more "worldly". They are beginning to take more control of their own destiny, including control of their health-care system. Perhaps most importantly, there are new economic initiatives on some Reservations. This is a key to breaking the cycle of factors that lead to the early mortality and excess morbidity among Native people.

For those working with the Native people, there are rewards too. While the job is very hard, and the effects sometimes invisible, the nurses and physicians who have worked in the SLZ can take great pride in what they have done. They truly have made a difference in the lives of many. That is a rare reward indeed.

"Quick Review"

Oliver Achleitner, 9T0

Cyberlog - Acute Respiratory Failure
192K, Dos 2.0 or higher, and a graphics adaptor
Cost: $695.00 U.S. per year
Published quarterly, each issue focuses on one primary care topic.

Available from: Cardinal Health Systems, Inc.
7562 Market Place Dr., Eden Prairie,
MN, USA 55344-3636
Tel: 1 (800) 328 0180

The programme consists of three basic parts: 1) Tutorials, 2) Case Studies, and 3) Tools.

There are six tutorials which go through various areas of basic respirology with the aid of graphics and user participation. In "normal V/Q Relationships", it shows how normal breathing affects PAO_2 and PaO_2 in addition to demonstrations of how to calculate PAO_2. It also allows the user to vary the amount of ventilation to observe changes in PAO_2, $PACO_2$ and PAO_2. In "Low V/Q Areas" tutorial, a graphical model is used with user participation to show the effects on blood gases when there is decreased ventilation of one lung. Intrapulmonary shunting effects on blood gases are shown graphically in the tutorial named "R to L Intrapulmonary Shunting". Effects of increased dead space on blood gases were also demonstrated in "Increased Dead Space". "Mechanical Ventilation I" and "Mechanical Ventilation II" demonstrate various pressure-flow relationships while the user can experiment with the parameters of a mechanical ventilator.

There are four patient case studies included with the program for the user to "treat". The options for management are given in multiple choice form. The user has the option of being corrected immediately after each step or at the end of the scenario. The corrections and their explanations are quite clear.

Along with the tutorials and case studies, the product also includes five tools for various respiratory calculations. "Predicted Pulmonary Function Test Values and Interpretation of Pulmonary Function Tests" provides a good graphical display of predicted and actual results. Under "Acid-Base Nomogram", a nomogram of acid/-base values was shown and the user is able to vary the the values to see which type of acid-base disorder exists at any particular pH, bicarb, and pCO_2 values. There is also a tool to calculate the A-a gradient called "Calculation of A-a Gradient". "Calculation of Wasted Ventilation" is done using three different methods: i) $PaCO_2$ and $PECO_2$, ii) VCO_2, $PaCO_2$, and VE, and iii) Weight, $PaCO_2$, and VE. "Calculation of Venous Admixture allow calculation of venous admixture of anatomic shunt by using measured blood gases.

All in all, this programme was easy to use and understand. Graphics were fairly poor but adequate and useful. The programme was only slightly less "boring" than a medical text. Although there is much room for improvement, Cyberlog - Acute Respiratory Failure meets its goal to be an educational aid for understanding the basic principles of respiratory physiology. It should be recommended as a good educational supplement in introductory respirology teaching.

Medi-Dict PC™

Ian Y. Pun, 9T0

Publisher: Physician Micro Systems, Inc., 801 E. Harrison St. Suite 105, Seattle, Washington 98102
Description: Wordprocessor program with template (fill in the blanks) capability. One program disk, one demo disk.
Hardware requirements: IBM PC, XT, AT or compatible, MS-DOS 2.0 or greater, 192K RAM or greater, two floppy drives or hard disk, IBM compatible printer
Warranty: 90 days with telephone support
Cost: $295.00 US

Traditionally, physicians establish routines for managing patients. Through experience, the history, examination, diagnosis and treatment become standardized but still allowing for individual variability. Therefore, patient notes become similar with only few major differences between patients. MEDI-DICT PC is a word-processing program which place the redundancies into user-defined "templates" in which the physician may then enter only the data that is unique to each patient.

MEDI-DICT PC is supplied on two diskettes, one program disk and one demo disk. Documentation is excellent as a 300= page manual in a loose-leaf binder is included. Installation of this program into your hard disk system is fairly fast and simple.

MEDI-DICT PC is a menu-driven program (in which you choose a command from a list of functions displayed) and pressing "?" usually provides instant help — a brief explanation of the commands available. The Main Menu provides options of editing and printing

patients' notes and "templates". The templates can be up to 600K or about 300 pages in length. Those who are computer proficient will learn all of the major features in about an hour. Unfortunately, every IBM PC program has its own unique definition of function keys and this program is no exception. Each function key has, at the most, three functions, depending on whether you press it alone or in conjunction with the shift or control key. Unfortunately, there were no keyboard overlaps nor stickers provided. Therefore it would be handy to keep the manual opened to the appendix for reference. The inconsistent use of the help key takes some getting use to. Outside the editor, help is "?". Inside the editor, it is "F2" and inside the actual help display (for more help), it is "F4". Nevertheless, the help functions are useful.

The user defines "templates" of patient notes by one of the options. For example, a common problem such as ankle sprain (see the example) can be entered into a template. After specifying the nature of the medical program and which "provider" you are (so that templates you use will be not mixed with those of other physicians as in a multi-practice situation), you enter the wordprocessing section. here you type out the text of the template, which could be a skeletal form of a patient progress note or lab result. Press the [prt sc/*] or [*] keys on the numeric keypad on a standard or extended keyboard respectively to enter places in the text where you would like to enter unique information in the future. The markers are denoted by a highlighted asterisk. "F1" saves the template for later use.

To create a patient note, you choose the transcribe option. You enter the patient's name and date. Then, you enter the wordprocessing section once again. Press "F6" to get a template. You freely edit this template to the nature of the patient's illness. Once you are satisfied with the note, you save it, exit the wordprocessing function and then print it. The asterisks shown on the screen will not be printed with the note. The print function allows several daily entries for batch printing (printing after accumulation of a number of documents) at a more convenient time. The print-out may be in a number of formats.

MEDI-DICT does the above functions quite efficiently and rapidly. Unfortunately, MEDI-DICT PC falls short of being a full-fledged wordprocessor. Although it provides the most rudimentary functions such as inserting, deleting, text justification, search and replace and block movement, it lacks the major functions of more sophisticated wordprocessing programs. There are no functions for mail-merging (combining a database of addresses with form letters to make "personalized" letters") as well as no facility for spelling check. it is not recommended for writing research papers because of the limited memory. However, you may save patient notes as standard ASCII text files for transferring to a more powerful wordprocessor if you desire these functions.

Perhaps the capability of storing notes on computer would tempt you to use this program for archiving patient records. The program is limited in the size of memory storage (only 50 patient notes can be stored on a floppy disk) and would be inadequate for the size of most practices. It is intended as an aid for medical dictation, not as database. You must file the printouts in a conventional manner. Its sister program, PRACTICE PARTNER (cost: $1995.00 US0, is intended as a database and can import files from MEDI-DICT.

MEDI-DICT's idea of templates would be useful in a practice where there are many redundancies, as in a routine laboratory or X-ray clinic. In conjunction with a tape dictation system, the elimination of repetitive phrases may reduce transcription time. Perhaps it is less useful in more varied general practices where templates are inefficient because you find yourself editing the entire template each time you write a note. Also, the idea of templates can be implemented on many general purpose wordprocessors although maybe not as efficiently. MEDI-DICT is specialized for a limited audience. You may find that it fits your present needs for simple medical dictation (it is more complex than an electric typewriter) but you may soon outgrow it and yearn for a more powerful system.

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Schizophrenia Gene Discovered

Zeke Milkovic, M.Sc., 9T0

Researchers at the University of British Columbia have located the gene region linked to schizophrenia. Schizophrenia is a psychiatric disorder that affects 1% of the population. It is a disease of complex etiology characterized by thought disorder involving pervasive delusions and hallucinations. Although schizophrenia clusters in families, the inheritance pattern is complex and does not follow Mendelian models. Specific gene involvement has not been proven.

Basset et al. (Lancet, April 9, 1988), studied a family of Asian descent in which two members have schizophrenia: a twenty year old male college student and his fifty-two year old maternal uncle. The twenty year old proband was noted to have different facial features from his parents and looked like his fifty-two year old uncle with schizophrenia, who also developed the disorder at age twenty. Because schizophrenia and subtle dysmorphic facial features coincided in these two men, they were examined thoroughly and found to have other physical abnormalities in common. Cytogenetic studies revealed that the two men had an identical unbalanced male karyotype with extra chromosomal material in the long arm of chromosome 1. Chromosome analysis of the mother, who is phenotypically normal, revealed that the chromosomal material came from a deletion of chromosome 5. Thus, she had a balanced insertion. Both the proband and his uncle have inherited the same derivative chromosome 1 from a balanced 1;5 insertion carrier parent along with a normal pair of chromosomes 5. Consequently, both are trisomic for the segment of chromosome 5.

This study provides the first link of schizophrenia to a specific region of the human genome. The use of DNA probes located within the candidate trisomic region of chromosome 5, will permit detection of restriction fragment length polymorphisms which coinherit with schizophrenia. To this end, a British research team led by Gurling, claims to have found positive genetic linkage with a marker in the chromosome 5 region. The report, which is to appear in Nature, included families with more than one member afflicted with schizophrenia. Thus, the finding may provide a means to identify which members of a family with a history of schizophrenia, carry the gene for the disease.

Small Intestinal Transplantation - A Closer Reality

Michael Weinberg, M.Sc., 9T0
Zeke Milkovic, M.Sc., 9T0
Z. Cohen, M.D., F.R.C.S.(C), F.A.C.S.

Surgeons at the Toronto General Hospital have been working towards successful small intestinal transplantation in humans. Patients suffering from major losses of their small intestine may become unable to consume food. Home parenteral nutritional support can be lifesaving, but longterm use is associated with many complications (especially in the paediatric population) as well as extreme cost - approximately $40,000 per annum.

In the 1960's transplantation of other organs was being perfected. However, small intestinal transplantation (SIT) has remained extremely difficult. The major obstacles to successful transplantation, are immunologic reactions associated with rejection or graft-versus-host-disease.

Prior to 1970, 7 attempts at SI allotransplantation were made, using conventional immunosuppression. All patients died shortly after transplantation. Thus the need for more experimental work was obvious. The advent of cyclosporine A dramatically changed the face of transplantation. The program in Toronto headed by Dr. Z. Cohen is one of the strongest and broadly based in the world. There have been many aspects of SIT studied. The major areas include organ harvesting, cyclosporine A pharmacokinetics and immunosuppression, allograft monitoring and tissue rejection. The basic techniques have thus been studied and worked out quite extensively.

These studies culminated in the SI transplantation of a 26 year old female, who had previously undergone subtotal colectomy and later proctectomy and ileostomy for Gardners Syndrome. Later a desmoid tumor was found and the entire small bowel was removed distal to the ligament of Treitz. Thus the patient was transplanted (1985) using cyclosporine A and methylpreduisolone for immunosuppression. This was the first small intestinal transplant ever performed using cyclosporine A. The patient's early post-operative course was satisfactory. On the 9th post-operative day the patient became deeply comatose, and died 2 days later. It was however felt that her demise was due to Cyclosporine A induced encephalopathy.

Despite the result of this case the investigators have a well developed protocol for small intestinal transplantation. Current research has been centred around the mechanism of rejection and the development of newer immunosuppressive agents which might produce less toxicity. Current plans still include proceeding with up to 5 further transplants. It is just a matter of time before success is achieved.

Human Sciatic Nerve Transplant

Catherine J. Brown, 9T0

The use of nerve autografts has become commonplace in reconstructive surgery but, until recently, peripheral nerve allografts were not considered possible as therapeutic alternatives. Earlier attempts at human nerve transplantation failed because the allograft was rejected by the host. However, the advent of immunosuppressive therapy has allowed Dr. Susan Mackinnon of the Sunnybrook Medical Centre and Dr. Alan Hudson of St. Michael's Hospital, Toronto, to perform a human sciatic nerve transplant on a young boy who was injured in a boating accident.

The boy suffered a 23 cm. deficit in his left sciatic nerve. Eight nerve fascicles were required to bridge this gap, necessitating the use of almost 2 metres of nerve had conventional autografts been attempted. Only 35-40 cm of nerve could be harvested from the boy to be used as an autograft. Thus, in order to avoid amputation, a sciatic nerve allograft was performed.

The donor and the recipient were matched by ABO blood group and the donor was tested to ensure that no CMV, HIV or hepatitis infection was present.

The recipient will be treated with immunosuppressive cyclosporin A and steroids for at least one year while the proximal stump of the sciatic nerve grows through the graft at a rate of 1 mm/day. Because the primary role of the allograft is to provide a conduit and suitable environment through which host axons can regenerate, short term immunosuppression may be sufficient to confer tolerance to the allograft.

One of the difficulties that may be encountered is the possibility that the host Schwann cells may be unable to completely coat the graft with myelin. This may be remedied by re-introducing cultured Schwann cells to the host. In the event that the distal leg muscles degenerate and become unresponsive to nerve impulses over time, free flap muscle transfers are a potential option to restore function to the leg.

A further five nerve transplants will be performed at St. Michael's pending a successful outcome of this first procedure.

Academic Aesthetic Surgery Service at Toronto Western Hospital

David Ewart, 9T0

While resident training in surgery generally follows a process of assisting in junior years with a progression to performing most surgical procedures in the senior years, aesthetic surgery has remained one area of plastic surgery where this process is difficult to adhere to. Generally, residents in plastic surgery are given relatively little opportunity to acquire experience in performing such procedures. Furthermore, they do not have the opportunity to be included in any preoperative consultations with patients, or in decision making processes regarding the appropriate surgical management of a patient. The reasons for this deficiency in training are perhaps understandable. Most cosmetic surgery patients have very high expectations of the post-operative outcome. As well, most aesthetic procedures are paid for by the patient directly. Furthermore, many procedures are performed under local anaesthetic. There is therefore a reluctance to allow residents, who are still training, to perform such procedures. As well, many of these procedures are now performed in private clinics not associated with resident training.

In light of this situation, an Academic Aesthetic Surgery Service was established at the Toronto Western Hospital in February of 1986. The service offers aesthetic procedures to patients at reduced cost (maximum of $200 plus the hospital costs). The initial consultation and all of the surgery is performed by senior residents, under the supervision of the staff surgeon. The revenue generated by the service is channeled entirely into teaching and research endeavours, through the Academic Enrichment Fund.

A post-operative evaluation of 52 patients was conducted between Feb/86 and Dec/87 by Drs. D. Ross, A. Freiberg, J. Taylor and M. Kara. Eighty-three percent of patients reported a high degree of satisfaction with the results, 12.3% reported a fair degree of satisfaction while only 4.7% reported extreme dissatisfaction. As well, post-operative complications were few.

It is interesting to note that over 25% of the patients evaluated were referred to the clinic by other plastic surgeons.

The clinic has therefore been recognized as a valuable teaching adjunct, while providing a useful service to the community.

BOOK REVIEW

Harrison's Principles of Internal Medicine (11th Edition) - Companion Handbook.

Edited by Eugene Braunwald, Kurt J. Isselbacher, Robert G. Petersdorf, Jean D. Wilson, Joseph B. Martin, and Anthony S. Fauci. McGraw-Hill, New York: 1988.

Oxford Handbook of Clinical Medicine.

By R.A. Hope and J.M. Longmore. Oxford University Press, Oxford: 1985.

Practical Guide to the Care of the Medical Patient.

By Fred F. Ferri. Mosby, St. Louis: 1987.

Ian Park, 9T0

You're doing your third G.I. clinic. You've just taken a history and done a physical examination. You think you know the diagnosis but you're not sure. You know you've read about this condition but your memory needs a boost — what is the differential diagnosis? What are the risk factors you should have elicited? What other systems can be affected? What other investigations should be done, and how should they be interpreted? What is the treatment?

The life of the medical student is filled with such uncertainties, (Well, mine is, anyway.) It is frustrating to tremble on the brink of connecting what you have previously learned with the patient you have just seen. And it is a little disappointing to have all your obvious errors pointed out by your clinician.

These three books have been written to help the student in such situations. Each is designed as a concise set of reminders for the harried student. The books are published in a small-size format so that they might be carried in a lab coat pocket (displacing, alas, otoscopes and sphygomanometers). And the authors have tried to arrange the contents in easy-to-use formats.

In the **Companion Handbook** to **Harrison's Principles of Internal Medicine**, the authors state that they "have now condensed the clinical portions of **Harrison's** into this pocket-sized **Companion Handbook** which residents and students can use on their trek through the inpatient, outpatient, and emergency services of a teaching hospital". (They also believe that it would be ideal to have a copy of the full **Harrison's** in one's pocket at all times, something I consider a mixed blessing at best, given its passion for excruciating detail.) The sequence of the sections in the **Companion Handbook** is generally similar to that in the main text, but, mercifully, the editors have not tried to write a simple condensation of that textbook. The focus is on the clinical aspects.

Unfortunately, the format of the presentations varies from chapter to chapter; ideally, one should be able to open the book anywhere and get the same sequence of information for any disorder. And, like the main text, this **Handbook** tends, in places, to have discouragingly long and detailed lists of things one feels one ought to know.

Of the three books reviewed here, this one is the one most like a textbook of internal medicine. For some readers, this is a good point, since it corresponds most closely to the way they have learned.

I have problems with this approach, however. In this book, the information is classified by organ systems, then by particular diseases. But in a clinical learning situation, you have to work "backwards" — from symptoms and signs to the diagnosis. Therefore, of these three books, I found this to be the least useful in a clinical setting.

Of the three books reviewed, this one has the most information; ironically, I found it frustratingly incomplete as I tried to use the book as a "mini-textbook". As such, it is not as useful as the full text. In summary, this book could be improved by re-organizing the information toward the clinical approach.

The **Oxford Handbook of Clinical Medicine** is more "user-friendly". The authors have been able to keep each topic to a single page; each facing page is blank so that one may enter notes that will be automatically indexed.

A second feature I liked is the relative uniformity of the format — here one can open the book to most pages and find the information presented in a standard sequence. This may be because there are only two authors of this book, unlike the six for the **Harrison's** handbook.

This book is the only one of the three to even attempt to rank the importance of the information. Subsections of each chapter are designated as "help for the houseman", "subjects for students", and "rarities for reference". Relevant pages in other sections are also noted at the beginning of each major section.

Finally, I found that the amount of information in each section was appropriate. There was no attempt to teach medicine one should have learned elsewhere; rather, there were lists of reminders to help one recall things like differential diagnoses and necessary investigations.

There were problems with this book, however. Since it is a British book, some of the terms used are unfamiliar to Canadian readers. (For example, FBC is a "full blood count", which I assume is the same as a CBC.') Secondly, like the **Harrison's** book, the handbook often assumes that the diagnosis is known; this this book too would be improved by a re-organization along clinical lines, so that the student might be aided in moving from signs and symptoms to diagnosis and disease.

I found the **Practical Guide to the Care of the Medical Patient** to be the most useful of the three books reviewed. Like the other two books, it is mainly organ-

ized by systems. However, this book has one critical extra: a section of differential diagnoses for 33 different signs and symptoms. From this, one can gain access to all the relevant sections in the rest of the book.

Within each section, there is strict adherence to the sequence of information given. This sequence is in a logical progression from etiology to treatment, similar to the sequence used in "real life".

More than the other books, this one is a collection of lists. I argue that this is the sort of thing one needs most in the clinical setting — that set of reminders of things that one is supposed to have learned before reaching the wards. For other readers, this would be the main weakness of the book — explanations are minimal. Thus, if one is not prepared, this sort of book can promote the worst in medical learning — the mindless memorization of disjointed facts. However, used with the proper background, this need not be the case with this book. It then becomes an excellent complement to the larger reference texts.

To try to test my subjective impressions, I invented seven questions of the type that arise in clinical teaching. I then rated how well I thought these questions were answered by the three books. (See the table which follows.)

	Question	Harrison's	Oxford	Guide
1.	Causes of an S_3	good	poor	poor
2.	Interpretation of pulmonary function tests	fair	fair	v. good
3.	Differential diagnosis of bloody stools	fair	fair	good
4.	Etiology of flank pain	poor	poor	fair
5.	Treatment of headache	good	good	poor
6.	Assessment of thyroid status	poor	good	v. good
7.	Emergency treatment of coma	poor	good	fair

In general, the results seem to confirm my previous impressions. This is, of course, a highly subjective test. But that is the nature of medical texts and handbooks —one man's meat is another man's poison. I strongly suggest that a prospective buyer make up a similar list of questions, then go to a bookstore to peruse the books before making a decision. Before investing in such a handbook, it is important to know one's own learning style and clinical requirements.

Finally, I leave you with the most important caveat: be sure that you actually need such a book before buying one. I have found that it is difficult to find the time to refer to such books during a teaching clinic — you are kept busy constantly. Until you find yourself needing such a quick reference (perhaps during Clinical Clerkship), I advise you to do without. It does make one's lab coat lighter.

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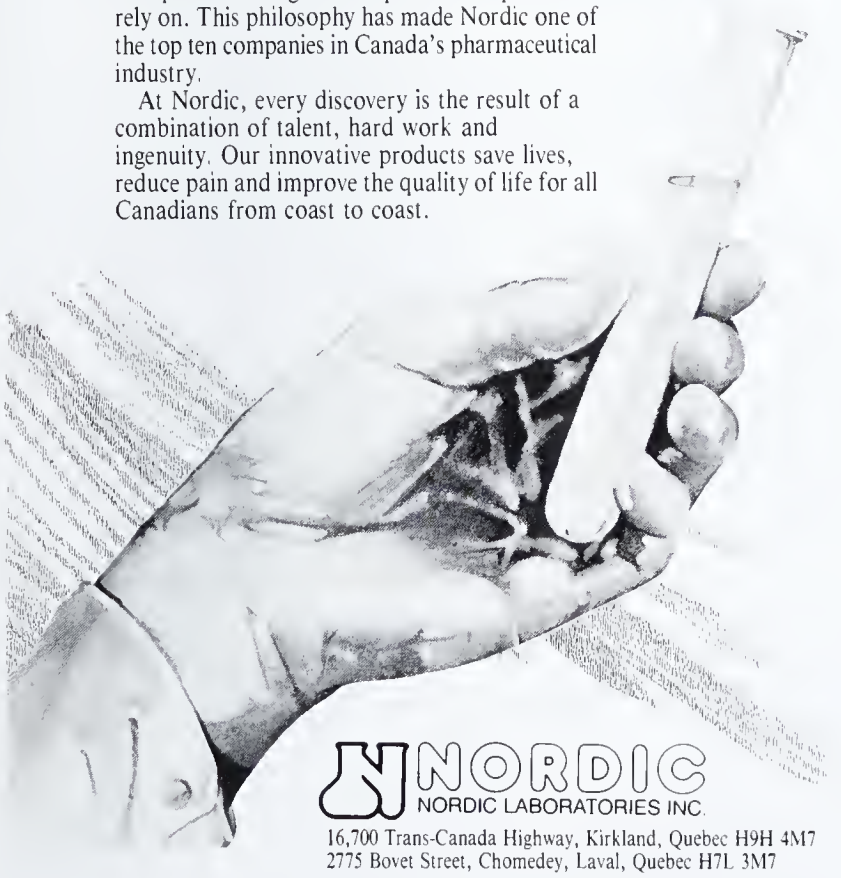

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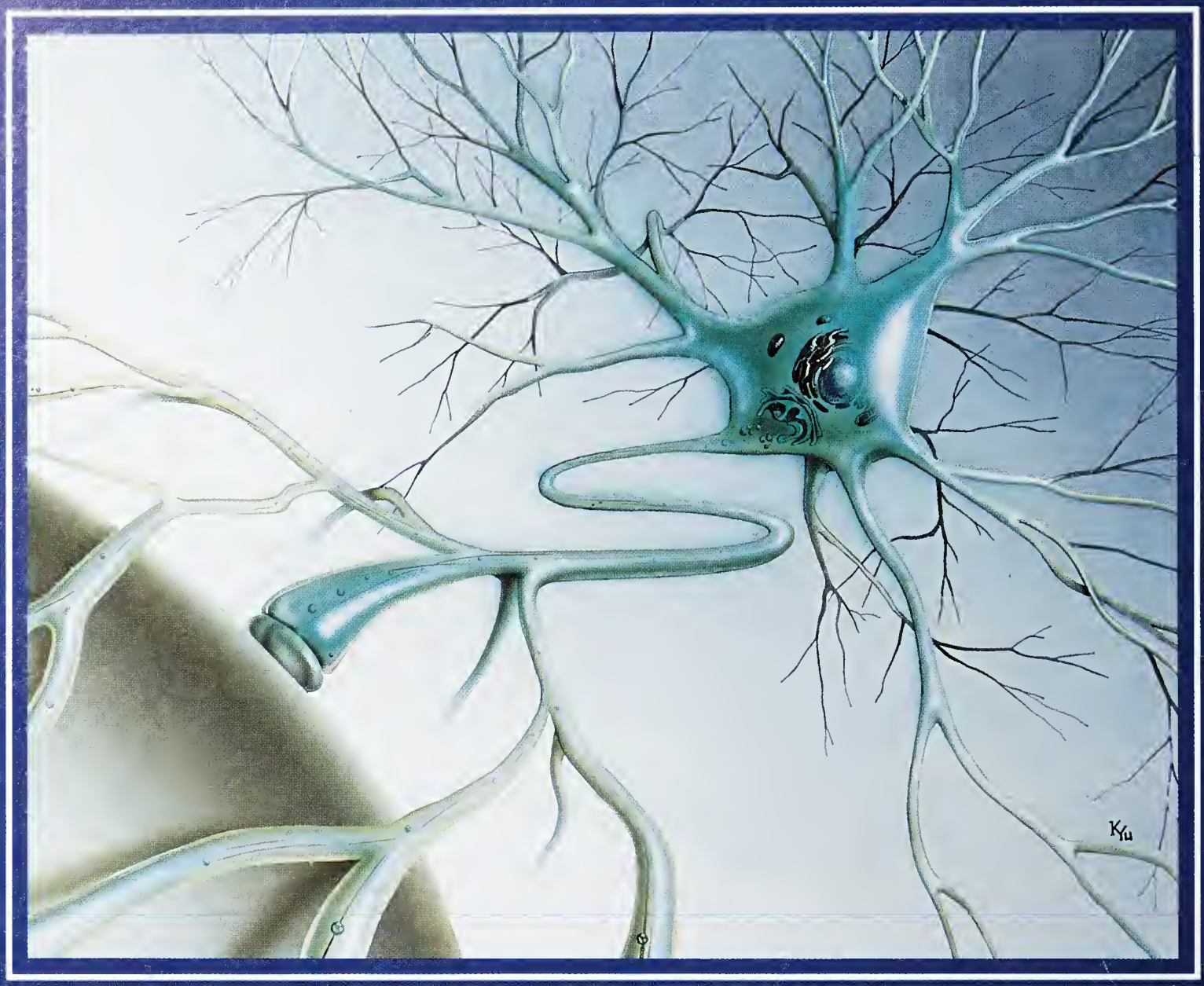

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